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THE PATHOLOGY OF CORONARY ISCHÆMIA.[1]

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BEFORE any discussion on the pathological basis of coronary thrombosis can be profitably followed a definition of the condition is necessary. Bedford (1939) pointed out that the clinical term "coronary thrombosis" is commonly employed to include all cases in which acute infarction of the heart is believed to be present, although in fact there may be no thrombosis of an artery demonstrable, but only an atheromatous stricture of an artery.

As some of the conditions examined in this paper do not always lead to a clinical picture of coronary thrombosis, but are nevertheless obviously related phenomena, I have preferred to use the term "coronary ischæmia" to cover all forms of defective blood supply to the myocardium, whether due to thrombosis, embolism or atheromatous change without actual blockage.

Although infarction of the myocardium was first recognized clinically early in this century, the study of disease of the coronary arteries and its associated clinical picture dates back to the middle of the eighteenth century. Jenner was at this period familiar with the association of *angina pectoris* and the hardening of the coronary arteries.

Reviews of large numbers of *post mortem* records show that coronary artery disease is common. In 1931, at the Presbyterian Hospital in New York,

[1] Read at a meeting of the Victorian Branch of the British Medical Association on July 5, 1939.

38% of all cases coming to *post mortem* examination were recorded as being cases of coronary artery disease (Levy, Bruenn and Kurtz, 1934).

Wright-Smith (1936) found in a series of coroner's cases in Melbourne that of 1,371 deaths investigated there were 339 from coronary obstruction.

In addition to these studies the use of the electrocardiograph in the elucidation of myocardial function in the human being has contributed to the clinical consciousness of this condition.

It is of great importance, therefore, to analyse the effect on the myocardium of the various changes in the coronary vessels, and also from this discussion to determine if possible the true meaning of the various electrocardiographic records obtained in these cases.

The study of coronary ischæmia will therefore require an investigation into the musculature, the blood supply of the myocardium, and an analysis of the changes in the myocardium following pathological changes in the vessels of the coronary tree. As an aid to the investigation of physiological activity of the musculature the electrocardiogram is of considerable use, and a brief discussion of the changes in the electrical activity of the muscle following coronary ischæmia is included within the scope of the paper.

Ventricular Muscles.

From time to time since Lower (1669) first described the superficial fibres of the heart anatomists have investigated the anatomical arrangement of the muscle bundles forming the ventricles of the heart. The basis of modern descriptions of these muscles is the work of Mall (1910), Flett (1927) and Robb (1934).

The following short account of the relationships of the muscle bundles and the associated diagrams is taken from a previous paper of mine (1939).

The superficial muscle layer of the heart arises from the tendinous structures at the base of the ventricles and passes around both ventricles as a left-handed spiral to the apex of the left ventricle. At the apex this layer of fibres forms two bands which wrap around each other as the vortex of the left ventricle. These bands then enter the inside of the left ventricle to form its inner wall and take part in the formation of the papillary muscles. It is possible by following the grouping of the surface fibres into the anterior or posterior band of the vortex to describe two muscle sheets in this superficial layer.

Superficial Bulbo-Spiral Muscle.

The fibres of the posterior horn of the vortex pass back from the apex to the base to reach the base of the ventricles around the base of the aorta and the left-hand side of the auriculo-ventricular ring. This portion of the sheet is known as the superficial bulbo-spiral muscle (Figure I). At the vortex the posterior horn enters the left ventricle, and the greater part of its fibres are continued towards the base on the posterior surface of the left ventricular cavity, passing directly into the auriculo-ventricular ring. Other fibres form part of the posterior papillary muscle and reach the auriculo-ventricular ring indirectly through the posterior papillary muscle. Although described as forming the posterior portion of the wall of the left ventricular cavity, they are rotating and start more on the left side and finish up on the medial aspect.

Superficial Sino-Spiral Muscle.

The other half of this spiral cone is called the superficial sino-spiral muscle (Figure II). It takes origin from the posterior and lateral surfaces of the auriculo-ventricular ring, and passes in a spiral fashion over the ventricles to form the anterior horn of the vortex of the left ventricle. Entering the left ventricle, it forms the lining of the left ventricular cavity chiefly on the anterior aspect, and also enters the anterior and portion of the posterior papillary muscles. These fibres also pass into the auriculo-ventricular ring directly and indirectly through the *chordæ tendineæ*, the two muscle insertions forming a complete ring on the inside in much the same way as the origins do on the outside. Between the outer and inner layers of these two muscles there are other muscle bands forming the bulk of the ventricles above the apical region.

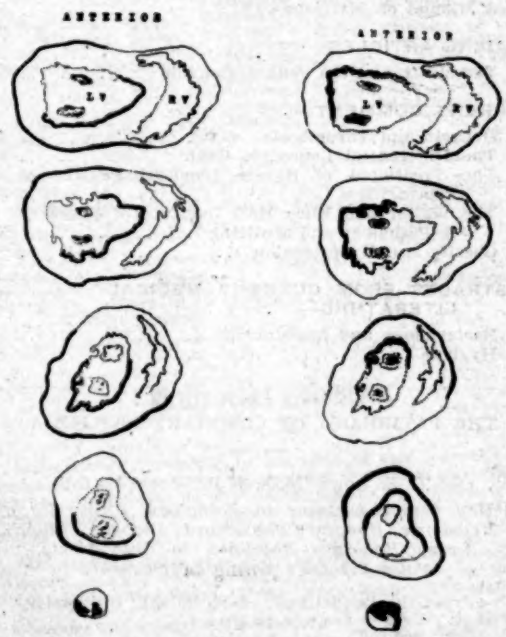

FIGURE I.
Drawing illustrating the superficial bulbo-spiral Muscle.[1]

FIGURE II.
Drawing illustrating the superficial sino-spiral Muscle.

Deep Bulbo-Spiral Muscle.

Arising from the left side of the auriculo-ventricular ring a band of fibres passes into the

[1] Figures I to V represent the extent of the particular muscle bundles described at a series of levels from apex to base of the ventricles, and are reproduced with the permission of the editors of *The Journal of Pathology and Bacteriology* from the author's paper on myocardial scars.

septum through the posterior longitudinal sulcus and forms a ring about the left ventricle around which the superficial and deep fibres wrap to form the vortex. It then returns to be inserted into the aortic ring in the region of origin. This band is known as the deep bulbo-spiral muscle (Figure III).

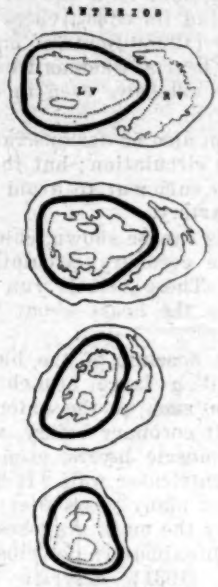

FIGURE III.
Drawing illustrating the deep bulbo-spiral muscle.

FIGURE IV.
Drawing illustrating the deep sino-spiral muscle.

Deep Sino-Spiral Muscle.

Arising from the anterior, lateral and posterior aspects of the left auriculo-ventricular ring another band of fibres can be traced passing across the posterior wall of the left ventricle and across the posterior interventricular sulcus into the right ventricular wall. As the band crosses the sulcus some fibres enter the septum, and these, together with those going around the ventricle, encircle it. In the region of the anterior interventricular sulcus the fibres pass deep into the right ventricle to enter the papillary muscles of the right ventricle. In their sweep they twist around some of the fibres of the superficial sino-spiral muscle and form the vortex of the right ventricle. The various branches of this sheet form the lining of the right ventricle and its papillary muscles. It is therefore inserted directly and indirectly into the right auriculo-ventricular ring. Other fibres continue across into the left ventricle and blend with deep fibres of the superficial bulbo-spiral muscle and take part in the formation of the papillary muscles of the left ventricle. This band is called the deep sino-spiral muscle (Figure IV).

Longitudinal Interventricular Muscle.

Arising from the aortic septum on its medial aspect is a band of fibres which pass downwards and forwards through the septum to the apical region. They blend with the fibres of the superficial sino-spiral muscle just before they enter the vortex, and are called the longitudinal interventricular muscle.

Scroll Muscle.

In addition to the above-described sheets of muscle fibres there is an extensive sheet of muscle which envelops the basal portion of both ventricles in the manner of a scroll. Arising from the tendon of the aorta on its medial aspect, it encircles the right ventricle, enters the septum from the posterior aspect and then passes through it to encircle the left ventricle external to the deep fibres of the superficial bulbo-spiral and superficial sino-spiral muscles (Figure V). It finally passes into the base of the heart amongst the insertion of the fibres of the superficial bulbo-spiral and sino-spiral muscles.

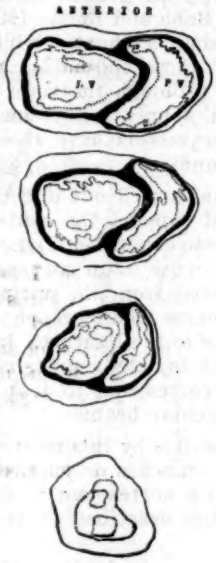

FIGURE V.
Drawing illustrating the scroll muscle.

Robb and Robb (1938) have recently determined the thickness of these various muscle bundles in a series of adult hearts, both normal and pathological. These hearts were examined in the diastolic phase.

They find that, throughout, the superficial muscles form a band about one millimetre in thickness. Normally in the right ventricle the deep sino-spiral muscle is about three millimetres thick, that is, 75% of the thickness of the wall. In the left ventricle the deep sino-spiral is usually three millimetres thick and the deep bulbo-spiral six millimetres thick, that is, the deep bulbo-spiral is normally 60% of the thickness of the wall.

In hearts with hypertrophy confined to the left ventricle the increase in thickness is mainly in the deep bulbo-spiral, where the proportion of the total wall thickness increases from 60% to an average of 68%, in some cases to 76%. In some of these cases

the actual thickness of the deep sino-spiral increases, but not in the same ratio.

It appears, therefore, that the deep bulbo-spiral is the important muscle in the maintenance of the left ventricular output. This agrees with the observations of Mall (1910) that it was difficult to demonstrate this muscle in children's hearts, but that it is easily seen in hypertrophied hearts.

Blood Supply.

By analogy with skeletal muscles, each of these muscle groups might be expected to have its own particular blood supply. The vessels concerned will run in planes alongside the muscle supplied, and their nutrient vessels will be derived from different parent vessels in different parts of the muscle.

This aspect of the coronary circulation has been neglected by nearly all writers on the subject, with the exception of Robb and Robb (1939) in Syracuse, who have published a chart, giving in detail the blood supply of these individual muscle bundles. They claim that although the main branches of the coronary system supply many muscle bundles in part, the smaller vessels have their field confined to one muscle bundle.

The author has carried out injection experiments which in general support the contentions of Robb and Robb. In these experiments uncoloured gelatine was injected into both main coronary arteries from cannulæ introduced from the aorta. At the same time some particular small branch was ligated and canalized distally to the ligature. Into this cannula blue gelatine was injected, under a pressure which was thought to correspond to that existing during life in that particular branch.

It has been possible by this means to demonstrate that such small branches do supply portions of the myocardium which correspond in distribution with the muscle bundles described (Figure VI).

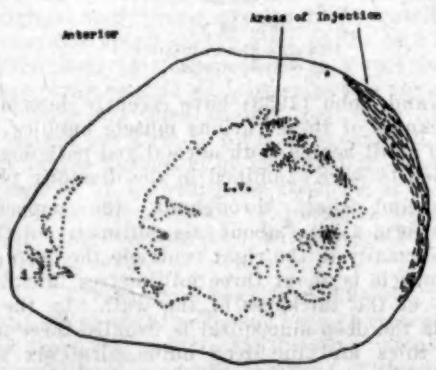

FIGURE VI.
Drawing illustrating the injection of gelatine into a single muscle.

It is well known that there are anastomoses by means of large arterial branches between different parts of the coronary circulation.

Most of the investigations on the anastomoses between the coronary branches have been carried out by injection experiments, and it is generally agreed that there are anastomoses between both the right and left coronary field and between arterial branches within the same field. There is, however, some divergence of opinion as to the extent of the arterial anastomosis and its effectiveness at different age periods. Gross (1921) found it more effective and general in older hearts, whereas I have shown (1937) that it can be extensive even in the child.

A capillary anastomosis can also be demonstrated between various parts of the circulation; but there is little evidence that this is sufficient to avoid an infarct after blockage of an artery.

Other anastomotic channels can be shown, chiefly in older hearts, between the coronary circulation and extracardiac arteries. These vessels run in the epicardial fat and leave the heart about the great arteries.

It will be seen from this account of the blood supply of the muscles that a large branch of one of the coronary arteries, such as the anterior descending branch of the left coronary artery, will supply portions of several muscle layers, even to the whole thickness of the ventricular wall. It has, of course, been recognized for many years that the areas of ventricle supplied by the main branches of the coronary tree can be approximately described.

MacCallum and Taylor (1931) describe the typical areas. For the anterior descending branch of the left coronary artery the infarct involves the anterior portion of the interventricular septum, sometimes going through to the right ventricle, and the apical and more anterior parts of the left ventricle. For the right coronary artery the field begins sharply in the mid-line of the interventricular septum, extends backwards behind the papillary muscles of the mitral valve to curve around on the more posterior part of the left ventricle. Scarring in its right ventricular field appears to be rare. For the circumflex branch of the left coronary artery the field is the median portion of the left ventricle between that of the descending branch of the left coronary and that of the right coronary artery. The septum is not involved.

Using again the analogy of skeletal muscle and its blood supply, we may say that blockage of one of the main branches of the coronary tree would be similar to blockage of the main vessel in a limb. In such a case one would not expect the resultant infarction of muscle to be confined to any one muscle, but to be seen in several separate muscles.

It is not surprising, therefore, to find that blockage of a main coronary branch involves portions of many separate muscle bundles.

In addition to the areas of supply of these main branches, it is possible to map out the region supplied by the smaller branches. This latter will, however, not be so accurate, for there is some degree of variation in the relationships of the various muscle bundles, and consequently of the areas of

supply in relation to the fixed points of the ventricles, although in regard to specific muscle bundles the nutrient supply may be reasonably constant (Figure VII).

Pathological Changes in Coronary Arteries.

Of the many pathological changes which may occur in blood vessels, atheroma and arteriosclerosis are the most important lesions affecting the coronary arteries. Although these conditions are separate, they are common and frequently occur together. To this composite condition the term "atherosclerosis" is often applied.

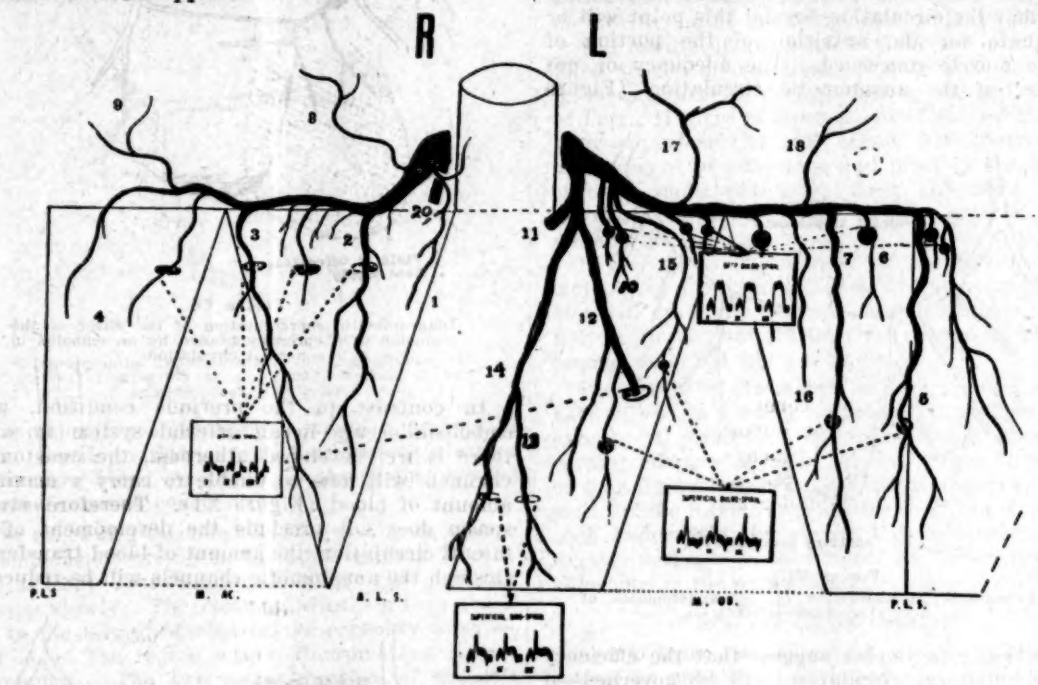

Figure VII.
Drawing illustrating the distribution of various coronary artery branches to different muscles. (From Robb and Robb.)

Atheroma.

Atheroma consists essentially of the deposition of cholesterol material in the deeper layers of the intima of an artery. This deposition, which may begin in the early years of life, is probably reversible in the early stages, but is associated later with proliferation of the intima. The deposit of cholesterol and the proliferation of the intima produce a plaque which encroaches on the lumen of the vessel. Ultimately atrophy of the media occurs.

In large vessels, such as the aorta, the presence of the plaque does not interfere with the blood flow; but in smaller vessels, of the order of the coronary arteries, the portion of the lumen blocked becomes appreciable and the blood flow is seriously reduced.

Sometimes in the late stages the endothelium covering the plaque may thin and the cholesterol material escape, leaving an ulcer on which clotting may occur. Rarely, hæmorrhage occurs into the plaque itself, producing a considerable increase in size.

Arteriosclerosis.

In contrast to atheroma, arteriosclerosis is essentially a change involving the media of the vessels. Commencing in many cases with hypertrophy of the elastic and muscle layers, it is only later that degenerative and fibrotic changes occur. As a result of the replacement of the contractile elements by fibrous tissue the vessels increase in length and being fixed at several points they must become tortuous.

The atheromatous change is chiefly confined to the main branches of the coronary vessels; the smaller branches running in the muscle planes are relatively unaffected. These smaller branches do, however, show the hyperplastic changes of arteriosclerosis.

Of the many associated factors which are incriminated as important agents in the production of arterial disease, *diabetes mellitus* deserves special mention. It is well recognized that arterial changes are common in individuals suffering from *diabetes mellitus* and it will not be surprising, therefore, that coronary lesions are relatively common. Warren (1938) found coronary sclerosis to be present in 96·7% of 509 *post mortem* examinations on diabetic persons and actual infarcts in 72 of 440 *post mortem* examinations.

Syphilis.

Syphilis, which produces arterial lesions in all organs of the body, is a relatively minor factor in

coronary artery disease, its commonest lesion being an intimal proliferation at the mouths of the coronary vessels in the aorta. This will occasionally produce complete obstruction.

Effects of Lesions in Large Branches of Coronary Arteries.

Embolism.

The effect of an embolus, whatever its nature, is completely to occlude the vessel in which it lodges, causing a cessation of any flow of blood into the peripheral parts of the arterial tree through that particular route. However, this does not necessarily mean that the circulation beyond this point will be inadequate for the nutrition of the portion of cardiac muscle concerned. The adequacy or not depends on the anastomotic circulation (Figure VIII).

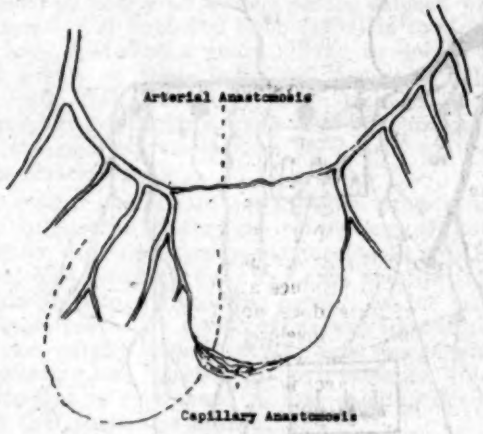

FIGURE VIII.
Diagrammatic representation of the anastomoses of the normal coronary circulation.

Injection experiments suggest that the efficiency of the collateral circulation will be governed—if there is no arterial disease—by the site of the block. In a previous paper I (1937) concluded that:

> The anastomosis may be adequate to inject the area obstructed if the obstruction is in the region of arterial anastomoses, although insufficient to fill completely the whole of the opposite circulation. When in the region of the capillary anastomoses it again appears to be insufficient to give a complete injection.

Before this conclusion about anastomoses in the injected specimens can be applied to the intact heart, the effects of the associated spasm of the arteries beyond the block must be considered. When an embolus lodges in an artery and suddenly occludes it, then spasm of the vessels beyond occurs, and this will necessarily interfere with the development of a collateral circulation (Figure IX). If, however, the complete block occurs slowly, spasm does not occur, and then possibly, if in the region of the arterial anastomosis, such an embolus will not produce infarction (Figure X).

Slow blockage of a vessel also allows a greater degree of collateral circulation to develop, because in addition to the absence of spasm there will be ample time for enlargement of the anastomotic vessels, which may be of insufficient size normally, even when spasm is absent, to carry a sufficient blood flow.

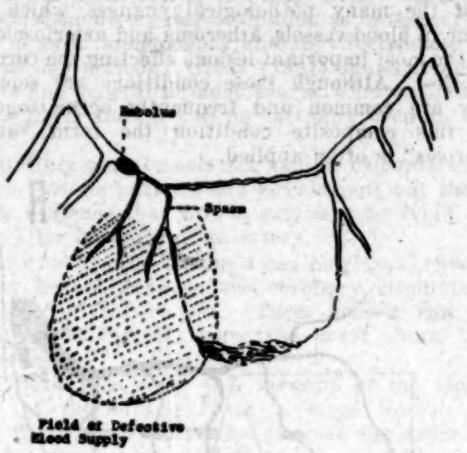

FIGURE IX.
Diagrammatic representation of the effect of the occlusion of a coronary branch by an embolus in a normal circulation.

In contrast to the previous condition, when embolism occurs in an arterial system in which there is well-developed atheroma, the anastomotic channels will now be unable to carry a maximum amount of blood (Figure XI). Therefore, even if spasm does not preclude the development of collateral circulation, the amount of blood transferable through the anastomotic channels will be reduced.

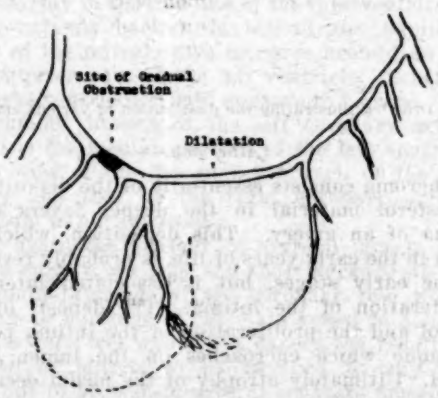

FIGURE X.
Diagrammatic representation of the effect of gradual obstruction of a coronary branch in a normal circulation.

Embolism of coronary arteries is a rare and usually fatal condition, but has been discussed in order to show the importance of vascular spasm and of atheroma in the development of collateral circulations in the coronary tree.

Thrombosis.

The frequency of the occurrence of thrombosis in coronary arteries is variously reported by different authors. Wright-Smith (1936) found it in only 45 of 495 cases of coronary obstruction, whereas Simpson (1939) recorded it in 330 of 580 cases. The opinion of most authors is, however, that thrombosis is a relatively uncommon event. It is so in my experience.

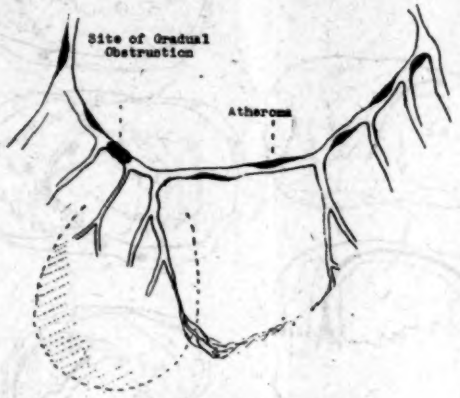

FIGURE XI.
Diagrammatic representation of the effects of gradual obstruction of a coronary branch in an atheromatous circulation.

The causation of thrombosis would appear to be either clot formation on an atheromatous ulcer or in the slow blood stream in an artery just beyond an atheromatous constriction.

The effect of a thrombus will be similar to that of an embolus occurring in an atheromatous circulation, except that the obstruction will probably develop slowly. The common sites for thrombosis are in the large branches of the coronary arteries, that is, in the region where atheromatous change is common. The two main branches of the left coronary artery seem to be especially vulnerable. The anterior descending branch has been called "the artery of sudden death". The relative incidence in the different arteries is shown in the accompanying table.

As a result of the blockage of these large branches, large areas of the myocardium will have either no blood supply or a very inadequate one. These areas will in general correspond to the fields of supply of these main branches.

The appearance of the infarcted area will vary according to the time elapsing after the occlusion. If sufficiently large, the infarct will interfere with the activity of the ventricles, producing abnormal rhythms and often sudden death. If this does not occur, then there will be an area of dead tissue which will undergo autolysis and be replaced by fibrous tissue. During the time of replacement the damaged region will be unable to stand any appreciable strain and so rupture of the ventricular wall may occur.

During and after the full development of scar tissue in the damaged area stretching may occur and aneurysm of the ventricular wall develop.

The results of an infarction are not always confined to the myocardium; if extensive, it usually involves either the pericardium or the endocardium or both. If there is involvement of the pericardium, then a pericarditis will ensue, with perhaps the development of adhesions and possibly the linkage, through small vessels in these adhesions, of the coronary capillary system with an arterial system outside the coronary field.

If there is involvement of the endocardium, then clotting on the damaged endocardium occurs. This clot will gradually become converted into a mass of fibrous tissue, but during the change pieces may become detached and act as emboli.

The effects of thrombosis in the coronary circulation will be to produce an infarct of greater or less extent. If this does not cause death, then myocardial changes develop, leading either to rupture or scar formation, with or without aneurysm of the wall. If the infarct is extensive it will be associated with endocardial or pericardial involvement, producing perhaps emboli from the endocardium or adhesions in the pericardium.

Ischæmia without Blockage.

Apart from the cases of coronary infarction caused by embolism and thrombosis, there remain a considerable number in which no complete blockage of a main branch can be demonstrated, but in which myocardial scarring is present.

In these cases apparently regions of myocardium have been deprived of their adequate blood supply either by spasm of smaller vessels or else by partial occlusion of the artery of supply and of its anastomotic branches. This partial occlusion occurs only in the larger branches; and whereas in these spasm appears to be unlikely, owing to their diseased

Author and Date.	Left Coronary Artery.		Right Coronary Artery.	Both Coronary Arteries.
Wright-Smith (1936)	38		6	1
Jervell (1935)	Left anterior descending branch 246 Left circumflex branch 22	268	70	21
Barnes and Ball (1932)	Left anterior descending branch 28 Left circumflex branch 17	45	20	—

condition, spasm can readily be imagined in the smaller branches unaffected by atheromatous change.

A complete block of a vessel is not necessary to produce these sheet scars. Barnard (1938) records a case in which such a scar occurred in an infant in whom the left coronary artery arose from the pulmonary artery. The scar was present in the area supplied by the venous blood. Maud Abbott (1927) describes similar scars in hearts with various congenital abnormalities in the distribution and origin of the coronary vessels.

Whatever the cause of the lack of blood supply, an infarct occurs. The area of infarct will, however, depend on the size of the vessel "blocked", and in many cases this will be one of the branches of a main coronary vessel, and so the infarct will be expected to be a portion of one of the ventricle muscles previously described.

Effects of Ischæmia in Small Branches of Coronary Arteries.

From the previous discussion it will be expected that any occlusion of a small artery will produce effects confined to one muscle bundle and probably to a part only of that muscle.

The study of myocardial scars which do not involve the whole ventricular wall has been neglected by nearly all writers. In 1874 Hilton Fagge described many of them and showed they were frequently sheets of fibrous tissue extending widely in planes in the ventricular muscle. Subsequent accounts do not accurately describe the extent of the scars.

Reconstructions of many of the scars show that they follow accurately muscle planes and correspond to portion of one or more of the muscles which have been described.

The following specimens illustrate these scars.

SPECIMEN 27.[1]—A sheet of fibrous tissue, resembling tendon microscopically, surrounds the left ventricular cavity. It occupies a mid-place in the muscle wall and extends as a complete cone from near the apex towards the base for one-quarter of the distance. This is well shown in the lower two drawings of Figure XII. At this region the cone becomes incomplete on its posterior aspect, but continues towards the base in the anterior and lateral walls of the left ventricle, and in the interventricular septum (see upper three drawings of Figure XII). In the lateral wall the scar gradually disappears, and it is only the antero-medial portion which passes to the auriculo-ventricular ring. The middle drawing (Figure XII) shows a marked thinning of the left ventricular wall on its anterior aspect half-way from the base to the apex. In this region the scar represents half the thickness of the wall. There is no aneurysmal dilatation present.

SPECIMEN 28.—The scar, occurring in the middle of the ventricular muscle of the left ventricle, extends from the apical region as a cone, incomplete on its lateral side. About one-sixth of the way from the apex to the base the scar splits into two bands (middle drawing, Figure XIII). They remain connected at the posterior end and pass to the right. One passes towards and almost reaches the subendocardial tissues of the right ventricle; the other passes towards the pericardial zone, ultimately coming almost to the surface. As these sheets are traced towards the base the endocardial portion appears in the interventricular septum close to the endocardium, whilst the pericardial one passes to the outer wall of the right ventricle in the mid-myocardial zone (upper drawings, Figure XIII). Two-thirds of the way from the apex to the base these sheets of fibrous tissue lose their posterior

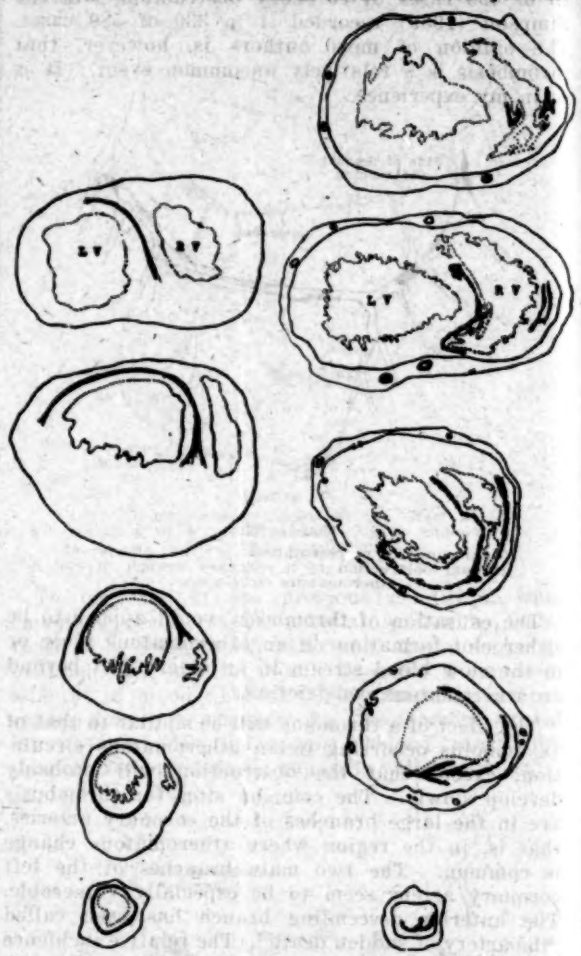

FIGURE XII.
Diagrammatic representation of the scar in Specimen 27.[1]

FIGURE XIII.
Diagrammatic representation of the scar in Specimen 28.

connexion and appear as two discrete bands, one in the interventricular septum and the other in the outer wall of the right ventricle. Both bands disappear four-fifths of the way to the base and do not reach the auriculo-ventricular ring.

SPECIMEN 31.—In this specimen there is a scar in the anterior wall of the left ventricle which is almost subendocardial. There is a layer of muscle a few millimetres thick between it and the endocardium. It is not seen in the septum, but extends in a sheet from the anterior end of the septum to a point about half-way around the lateral wall of the left ventricle. It extends from apex to base, as is seen in the drawings of Figure XIV.

SPECIMEN 63.—This specimen shows two scars which appear to unite near the apex of the left ventricle. One scar is almost subendocardial and the other deep in the muscle of the wall (Figure XV).

[1] The numbers of the specimens are the serial numbers of a large series of hearts examined in this and other investigations.

[1] Figures XII, XIII, XIV and XVI are reproduced with the permission of the editors of *The Journal of Pathology and Bacteriology* from the author's paper on myocardial scars.

The subendocardial scar is a sheet extending from the apex of the left ventricle to a point about three-quarters of the way to the auriculo-ventricular ring. It covers the posterior surface of the ventricular cavity in the lower portions, but gradually disappears from the lateral aspect as it goes towards the base, until finally it is seen only on the septal aspect.

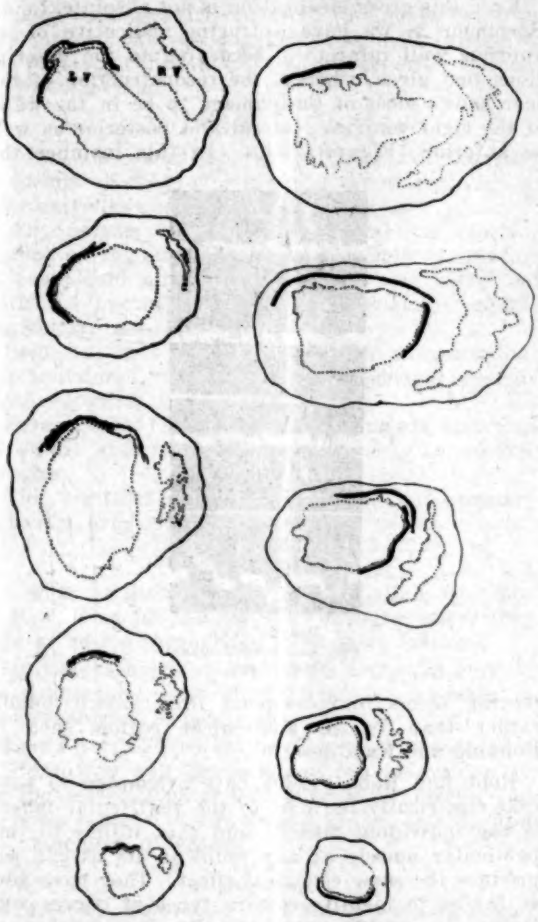

FIGURE XIV.

Diagrammatic representation of the scar in Specimen 31.

FIGURE XV.

Diagrammatic representation of the scars in Specimen 63.

The deep scar appears to blend with the superficial one at the lower end of the interventricular septum. When the scar is traced towards the base from this point, it diverges from the subendocardial one and is seen in the septum and anterior wall of the left ventricle, separated both from the subendocardial scar and the surface by muscle bands. In this region there is some thinning of the ventricular wall (middle drawing, Figure XV). The scar is not seen in the septum beyond a point half-way from apex to base, and it disappears from the anterior wall seven-eighths of the way to the base. There is also considerable thickening of the endocardium to be seen on the anterior aspect of the left ventricular cavity.

In this specimen the lower half of the anterior descending branch of the left coronary artery was completely occluded by atheromatous change.

These scars can be closely correlated with the distribution of separate muscles.

Number 27 corresponds to portion of the deep bulbo-spiral muscle.

Number 28 corresponds to portion of the deep sino-spiral muscle.

Number 31 corresponds to portion of the superficial sino-spiral muscle.

Number 63: the deep part corresponds to portion of the deep bulbo-spiral muscle, the subendocardial part to the superficial bulbo-spiral muscle.

As these scars correspond to portions of muscle bundles, they cannot be due to occlusion of a main coronary branch, for they extend beyond the field of any one main branch. It is also difficult to explain them by a lesion of a small branch, for, whilst its distribution may result in a lamina of muscle destruction, a single small branch obstruction would give only a portion of the scar seen in most of the specimens described.

In only two of these cases, numbers 27 and 28, is a clinical history available.

Specimen number 27. In this case the patient was a male, aged about sixty years. He was admitted to hospital for operation on a carcinoma of the colon. He gave no history of any existing or previous cardiac disability. He died from post-operative sepsis.

Specimen number 28. F.W., a female, aged sixty-two years, had had nocturnal dyspnœa for months. An acute condition, diagnosed clinically as coronary thrombosis, occurred twelve days before death. An electrocardiograph tracing taken six days before death suggested an anterior wall infarction. Death was clinically due to a further coronary thrombosis.

The clinical history of number 27 is important, for it shows that these scars may develop in extensive areas of muscle without clinical symptoms. This is a common occurrence, for Simpson (1939) records myocardial scars in 1 in 30 of all autopsies. Further, in his series of 93 instances of myocardial scars there were 82 cases in which there was no history of previous cardiac disability.

Apparently, therefore, some areas of cardiac muscle can become functionless without marked interference with cardiac action.

Robb (1939) in a recent article analyses the importance of infarction in the various muscles and concludes that injury to the superficial muscles is relatively unimportant. Injury to the deep sino-spiral is, experimentally, often fatal, for this muscle does the work of the right ventricle and some of the work of the left. The deep bulbo-spiral is of less importance than the deep sino-spiral, for it is confined to the left ventricle; but in hypertrophic hearts it occupies a large proportion of the musculature of the left ventricle and is relatively increased in importance.

Electrocardiogram.

It will be obvious that any interference with the blood supply to the cardiac muscle sufficient to cause death of muscle cells will interfere with all of its activities, not merely with its ability to contract. One of the attributes of cardiac muscle, or perhaps of the closely associated Purkinje fibres,

is conduction of the impulse which is the stimulus to muscle contraction.

The electrical changes in the heart which are recorded in the electrocardiographic tracing are due to this impulse passage and not to the actual muscle contraction. It has often been assumed that the electrical effects are due to muscle contraction because the two events are so closely related and occur usually, but not always, in sequence. It has frequently been demonstrated that an electrocardiogram can be obtained from hearts in which muscular activity has been arrested by various means (Weber 1926, Klewitz 1916, Henrijean 1925).

If this is the basis of the electrical change recorded by the electrocardiograph, then a priori it is extremely unlikely that a single tracing will give any information about the nature of the pathological condition which has produced the change in the heart muscle. It can demonstrate only a variation in the normal electrical activity of the muscle. It will not distinguish between inflammatory involvement, vascular occlusion or even trauma. In addition to this difficulty in the interpretation of the electrocardiogram in terms of pathological change, we cannot assess the relative parts played by the currents of injury from the damaged muscle and the effect of the deviation of the path of the impulse through the cardiac muscle. The fact that the electrocardiogram after an infarction has healed is different from that recorded before the catastrophe, is taken to indicate that the path of the impulse does change. The rapid variations that occur from day to day in the tracings taken immediately after a coronary occlusion are said to be due to the variations in the currents of injury from the damaged region. Further, as the pathological process heals, the electrocardiogram will return towards or even to normal. Hence the electrocardiogram may not give any information concerning past damage to the myocardium.

The change in the electrocardiogram in cases of ventricular dysfunction will be in the *Q R S-T* complex, and predominantly, experience shows, in the interval between the *Q R S* and *T* complexes. Instead of the tracing in this portion being isoelectric, it is displaced above or below this axis in one or more leads. This is the tracing described by Pardee (1920) as characteristic of coronary thrombosis. It is not surprising that the change may be seen in some leads only, for the recording of the electrical change is being made at a distance from the source of the electrical disturbance. In each lead there will be only that component of the electrical potential which is in that particular plane. The use of three leads in planes at right angles has been found the most satisfactory method of analysing these changes. These leads are Lead I, Lead III and Lead IV.

It has been generally accepted that these changes in the tracing are characteristic of ventricular muscle dysfunction. Many attempts have been made to correlate specific changes in the tracing with specific anatomical regions of the ventricular muscles. Bohning and Katz (1938), reviewing the literature, have shown that most authors consider infarctions of the anterior wall of the ventricles to give an elevation of the *R-T* interval in Lead I and to depress the *S-T* interval in Lead III, and that this change is reversed in posterior wall infarctions.

Even this gross localization is not absolute, for in Specimen 28 we have a tracing indicative of an anterior wall infarction, according to the descriptions just given, whereas the reconstruction of the scar shows most of the damage to be in the walls of the right ventricle, lateral and posterior as well as anterior (Figure XVI). In this instance the

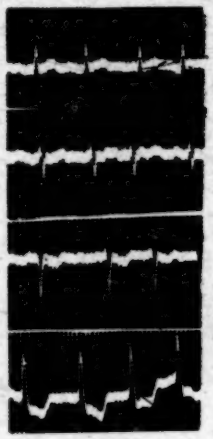

FIGURE XVI.
The electrocardiogram from Case 28.

tracing seems to correspond to a muscle bundle rather than to an anatomical region such as Bohning and Katz describe.

Robb and Robb (1939) have attempted to show that electrically the unit of the ventricular muscle is the individual muscle, and that injury to that particular muscle at any point in its extent will produce the same electrical effect. They have gone so far as to identify certain types of curves with lesions in specific muscles, and maintain that all curves of muscle damage in the ventricles are composite tracings of the specific ones.

In addition to the effects on muscle activity just described, two other types of change are met with in coronary occlusion. First, involvement of the conducting system in its main branches will produce partial or complete dissociation of the auricular and ventricular rhythms—partial or complete heart block. Secondly, the injury to the muscle may alter its irritability locally and give rise to ectopic ventricular beats—extrasystoles or even ventricular fibrillation. Occasionally auricular fibrillation is seen, but this will probably be due to auricular muscle involvement.

From this discussion we may safely conclude that while the electrocardiogram can give evidence of ventricular muscle dysfunction, we cannot at present interpret it in terms of pathological con-

ditions. There is in addition some empirical evidence enabling a limited localization of the site of injury to be determined.

If functional independence of the various muscle bundles can be established, then possibly we shall be able to localize more accurately the site of damage and perhaps venture a prognosis, based on the muscle involved, together with a knowledge of the pathological change obtained from other methods of investigation.

Summary.

In a discussion on the pathology of coronary ischæmia an account is given of the musculature of the ventricles and of its blood supply.

An analysis of the effects of various vascular lesions on the efficiency of the coronary circulation is considered and the end results are correlated with the known physiology and anatomy of the ventricular muscles.

Some evidence is produced which indicates that the individual muscles have independent specific blood supplies.

Several cases of myocardial scarring are described in detail and related in distribution to specific muscles.

The electrocardiographic changes in coronary ischæmia are briefly recounted.

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I wish to thank Professor MacCallum and Mr. E. S. J. King for the help and encouragement they have given me throughout these investigations.

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SOME PRACTICAL CONSIDERATIONS IN CORONARY OCCLUSION.[1]

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So much has been written regarding the classical manifestations of coronary occlusion that it is redundant to attempt to make a complete survey of the points of historical interest or to present a detailed account of the clinical, pathological, electrocardiographic and skiagraphic findings; and the majority of physicians have their own particular routine for treatment in these cases. It is, however, always of value to reconsider the pathology of disease, and to correlate the known pathology with the clinical manifestations of disordered physiology. I propose, therefore, to present for consideration: (i) the types of lesions in the coronary circulation, (ii) the effects of ischæmia on the cardiac musculature, (iii) the clinical types of coronary disease, (iv) the clinical indications of occlusion of the coronary circulation, and (v) some points in diagnosis, prognosis and treatment.

The Lesions in the Coronary Arteries.

The pathology of the condition has been described by Wright-Smith (1936), who states that the commonest lesion in the coronary arteries in instances of occlusion is atherosclerosis, which, although it may suddenly cause symptoms, is gradual in its development. The lesion is not confined to the coronary arteries, but is part of a general systemic affection, although the maximum evidence of the disease may be in the coronary

[1] Read at a meeting of the Victorian Branch of the British Medical Association on July 5, 1939.

arteries. Levine (1929) pointed out that the pre-existing "hypertension is probably the most common single etiological factor in the development of coronary thrombosis". (He classified all cases of occlusion as coronary thrombosis.) There is always the difficulty that the patient may not have had a previous knowledge of hypertension, and it must be remembered that after coronary occlusion has occurred clinical evidence of atherosclerosis may not be elicited. Atherosclerosis undoubtedly does occur without accompanying hypertension, which further complicates the interpretation of clinical findings.

Thrombus formation was present in 23 of Levine's 46 cases that came to autopsy, and in every case the thrombus was attached to an underlying arterio-sclerotic process. Simpson (1939) is reported as stating that he found 330 instances of thrombosis in 580 deaths from coronary disease. On the other hand, Wright-Smith (1936), whose method of examination is described and whom many of us have seen at work, found thrombus formation in only 45 of 495 patients, and points out that it is not always the terminal event.

Embolism is a rare cause of infarction. Syphilis alone, although a common cause of narrowing of the orifices of the coronary arteries, is generally acknowledged to be a rare cause of coronary occlusion. Other associated conditions, such as diabetes, gout and anæmia, are contributory factors because of their effects in aggravating the arterial change or increasing the anoxæmia of the cardiac musculature.

These lesions provide an adequate explanation for the changes in the cardiac muscle; but what of the symptoms? *Angina pectoris* has been variously attributed to lesions in the aorta (Clifford Allbutt, Wenckebach), fatigue of a weakened myocardium, comparable with the muscular pains of cramp (Mackenzie), and narrowing of the coronary arteries (originally proposed by Jenner). Boyd (1931) strongly supported the theory that "angina is the cry of pain of a diseased aorta"; but the extensive work which has been done in connexion with occlusion of the coronary arteries definitely indicates that muscular ischæmia, absolute or relative, is the most satisfactory explanation of the pain. When a coronary artery is acutely obstructed pain results which is anginal in character and distribution. Pain of a similar nature is produced when the muscles of a limb are exercised after the application of a tourniquet, and it is most "probable that anginal pain is caused by a specific chemical or physico-chemical change in the musculature of the heart, and that this is usually, if not always, consequent upon defective blood supply" (Lewis, 1933). The conception of ischæmia includes not only change in the coronary artery, but the state of tone of coronary artery and arterioles; it allows that spasm of the coronary arteries may cause or contribute to a deficient blood supply, which may be made good by vasodilatation; and it helps in the understanding of *angina pectoris* in anæmia.

The Effects of Ischæmia on the Cardiac Musculature.

The pathological changes in the cardiac muscle resulting from persisting ischæmia are either fine fibrosis or areas of infarction. The former appears to be a result of occlusive changes in the small branches of the coronary arteries and may be detectable only on microscopic examination or may be evident macroscopically. Infarction on a large scale was found in only 87 (17·6%) of Wright-Smith's series, whereas all of Levine's 46 cases showed infarction. Since a clinical diagnosis had been made prior to death in Levine's series, this group is not comparable as being a selected series of cases, and it can safely be assumed that many cases of occlusion occur without gross infarction.

The work of Robb and Robb (1938), which has been followed up by Dr. T. E. Lowe, has very great implications which cannot as yet be applied with certainty; but the additional evidence which he has produced is indicative of the importance of detailed study of the distribution of cardiac lesions resulting from ischæmia. It remains to be seen whether there is any relationship between the site of the occlusion, the muscle bundles involved as a result thereof, and the occurrence of pain.

Clinical Types of Coronary Disease.

Ischæmia of the cardiac muscle appears to be the most satisfactory explanation of the cause of anginal pain, and yet it is clear that fibrosis occurs in the myocardium without there being any clinical evidence of ischæmia. The occlusive process is slowly progressive and the pathological changes may be very widespread and result in gross myocardial insufficiency without the occurrence of pain. Actual infarction may be painless, and Levine refers to occlusion of the right coronary artery without chest pain.

In Wright-Smith's series, in 72% of the 339 cases examined for the coroner there was no past history of any symptoms severe enough to make the patients seek medical attention. In the hospital series precordial pain was recorded in only 39%, but dyspnœa was present in 68% of the 156 cases. I suggest that the more gradual the occlusion, the greater the likelihood of a painless course.

Clinically it is useful to consider anginal pain in three categories: (i) angina of effort (Heberden's angina), (ii) spasmodic angina, (iii) acute coronary occlusion.

Angina of effort and spasmodic angina do not enter into the scope of this paper, excepting in so far as they are concerned in the differential diagnosis, and the treatment is essentially different from that necessary in coronary occlusion; but it must be remembered that angina of effort is a frequent precursor of and a common sequel to an attack of coronary occlusion.

Clinical Manifestations of Coronary Occlusion.

A diagnosis of coronary occlusion is made from consideration of the history, the result of physical examination and the electrocardiogram.

Symptomatology.—The pain occurring in complete coronary occlusion is sudden in onset, usually severe, and associated with a sense of constriction. Most commonly thoracic in distribution, it is referred to the substernal region with variable irradiation across the chest, into the neck and jaws, over the epigastrium, to the upper limbs, more often the left, even to the back. There may be a sensation of choking or constriction in the throat. The patient, if he has previously suffered from *angina pectoris*, will describe this as a similar pain but much more severe, or recognizes it as a different type of attack. The pain is variably described as a feeling of severe oppression, crushing, boring, or as though the chest was in a vice; a stabbing pain is, for practical purposes, not cardiac in origin. The pain lasts hours or days instead of minutes, and is unrelieved by vasodilators, such as trinitrin and amyl nitrite; and even large doses of morphine injected subcutaneously may give only slight relief. In other cases the pain may be merely a dull substernal aching or gnawing sensation. Associated with this pain there may be stabbing pain, pleuritic in origin; and I have seen patients in whom pain was pleuritic in nature and subsequently shown to be due to pleurisy; but the distress and sense of oppression were present and indicated the underlying lesion. In one instance there was pain on top of the left shoulder and in the left lower thoracic region and upper part of the abdomen, apparently diaphragmatic in origin.

Restlessness is common, so that the patient cannot lie down or be quiet; and the pain is often not materially affected by exercise and is unrelieved by rest.

Distress may be due to the intensity of the pain, to urgent dyspnœa, or to both. Orthopnœa may be pronounced. Vomiting sometimes occurs at the onset of an attack, and when it is associated with abdominal pain an acute abdominal crisis is simulated. In contrast to this picture we have that presented by patients who have had no pain and who present themselves with congestive failure or after embolism has occurred, and in whom typical electrocardiographic tracings of cardiac infarction are found. Sudden death may occur without any premonitory distress.

Physical Signs.—There is a variable degree of shock and circulatory collapse with subnormal temperature; some patients actually lose consciousness. The skin is pale, moist, sometimes ashen grey in colour. The pulse tends to be rapid and of small volume; but in Case III the patient had a slow heart rate owing to complete heart block, which occurred within twenty-four hours of the onset of the pain. The blood pressure usually falls rapidly in association with the shock, but there may be little alteration or a slow fall in blood pressure over a period of days. Signs of congestive failure with œdema at the bases of the lungs, distended veins in the neck, hepatic enlargement and œdema of the limbs and loins, may develop rapidly or slowly or they may not appear at any stage. Adventitious sounds are frequently heard over the lungs, and an attack of acute pulmonary œdema may develop concurrently with the pain.

Physical examination of the heart at this stage may reveal no abnormality, or merely some cardiac enlargement due to preexisting hypertension; but it is essential to reexamine the precordium at frequent intervals in order to detect alterations in physical signs and disturbances of rhythm. In the patient with heart block, already mentioned, the heart sounds suggested the possibility of this rhythm. The heart sounds are often distant and muffled. Gallop rhythm may be present. In the absence of aortic disease acute left ventricular failure should suggest coronary occlusion.

After subsidence of the acute attack the blood pressure tends to remain at a fairly constant level; but the level of the blood pressure bears no relation to the immediate prognosis. There is an early slight rise in temperature, which may last for from one to several days, may rise to 101° or 102° F., but which is more often in the region of 100° F. It may be necessary to take rectal temperatures to detect this rise. A leucocytosis of 12,000 to 15,000 or exceptionally up to 25,000 per cubic millimetre accompanies the fever. These changes are probably directly attributable to the infarction, for the occurrence of sterile infarction in other parts of the body is frequently accompanied by fever and leucocytosis.

Pericarditis, when it occurs, is valuable confirmatory evidence of infarction. Levine states that only 13·8% of patients showed this sign; but I am confident that it will be found more often than this if frequent examinations are made. I have found the typical to-and-fro pericardial friction rub to be present for only a few hours, and have detected it only at one examination of the patient, so that it would be very easy to miss. The rub may be localized to a small area or widely diffused, and has no characteristic features. It is not associated with pain. A pleural rub can be differentiated by its different quality, relation to respiration, and characteristic stabbing pain. Possibly the result of pulmonary infarction, the presence of a pleural rub may cause some difficulty in diagnosis, especially if it develops early in the illness and there has not been pronounced pain indicative of coronary occlusion. The pleural rub may be localized to the region of the apex beat, which causes an added difficulty.

Other Associated Manifestations.

An uncommon associated condition is acute mental confusion with disorientation for time and place, which, developing during the illness, is very disturbing to the clinician and to the relatives of the patient. Complete recovery can take place, the improvement being gradual and commensurate with improvement in the circulation.

Embolism in either the pulmonary or systemic circulation is a direct result of detachment of an intraventricular thrombus. Albuminuria and

abnormal formed elements may be present in the urine quite apart from embolism.

Course.

The course of the illness is exceedingly variable and must be considered as that of cardiac failure with or without congestion, in which there is a peculiar liability to sudden death from ventricular fibrillation or cardiac rupture, to abnormalities of rhythm, to embolism, to rapid or slowly progressive circulatory failure, and to a varying degree of recovery with or without angina or congestive failure. Recurrences are common at intervals varying from a few hours to many years.

Electrocardiography.

Parkinson and Bedford (1928) pointed out the successive changes in the electrocardiogram after cardiac infarction. These may briefly be described.

1. The *R-T* deviation. This is evident during the first week or more after infarction, and its occurrence is coincident with myocardial necrosis. It is usually best seen in Lead I and Lead III with elevation in the former and depression in the latter, and while most often the deviation is from one to three millimetres, there may be up to six millimetres of deviation. While usually transient, it may persist, and *T* waves are often less evident when it is pronounced. In Case IV there was pronounced deviation only in Lead IV at an early stage after infarction.

2. *T* wave changes. These are fully developed in two to three weeks, and posibly indicate impaired myocardial function. There are two main types: (*a*) *T* 1 type with a negative *T* in Lead I, the *T* wave being deep and sharply inverted, the coronary *T* wave or Pardee curve. *T* 3 is usually positive and pointed. (*b*) *T* 3 type with sharply pointed inversion in this lead, often a deep *Q* wave, and *T* 2 inversion, which is more commonly associated with it than with the *T* 1 type. (*c*) Atypical *T* waves, which may remain upright.

3. Changes in the initial ventricular deflections (*Q R S*). A low voltage is common; widening, often with notching, may be present, and evidence of bundle lesions may be found.

The *T* 1 type has been held to indicate anterior lesions, and the *T* 3 type posterior infarction of the left ventricle.

Infarction of the lateral wall of the left ventricle has been shown by Wood, Wolferth and Bellet (1938) to produce electrocardiographic changes which can readily be missed. *R-T* depression is found in Lead IV and usually is present in Leads I and II. If the chest electrode is placed at or to the left of the apex beat the deviation in Lead IV is most evident. This type of infarction is easily missed, because auricular fibrillation is frequent, digitalis may produce the same effect and signs may disappear rapidly and completely from the electrocardiogram.

It is worthy of note that Bourne and Evans (1938) found that 17% of their series of 80 patients with *angina pectoris* examined electrocardiographically showed evidence in Lead IV only. They considered that Lead IV helped to emphasize the importance of slight changes in Leads I, II and III; but in one-third of the cases there was no change in the electrocardiogram. In tracings taken during and immediately after an attack of anginal pain occurring in one of my patients the only difference to be noted was an increase in voltage after the attack.

Localization of cardiac infarcts in man has hitherto been determined by the *T* 1 and *T* 3 types of curve and described as anterior and posterior respectively; and infarction of the lateral wall of the left ventricle may be detected by Lead IV. Robb and Robb (1939) are not satisfied with this method of localization and have put forward evidence in favour of a muscle-bundle mode of localization while admitting that there is essential agreement between topographical (anterior-posterior) and specific (muscle-bundle) methods. They conclude that:

> As a "thumb nail" diagnostic method one may ignore Lead II and note that *R-T* 1 depression indicates involvement of the S.B.S. muscle;[1] if *R-T* 3 is depressed, the D.S.S. is implicated; an S.S.S. lesion raises both *R-T* 1 and 3 above the iso-electric level. *R-T* take off from the peak of *R* (or near the peak) in all leads, especially in the presence of low voltage, indicates a D.B.S. lesion. Inequality of the *R* bases is to be sought and its presence is more instructive than its amplitude.

Combined lesions often occur, the most frequent combinations being S.B.S. and D.S.S. giving iso-electric *R-T* waves, and S.S.S., S.B.S. and D.S.S. with elevated *R-T* 1 and depressed *R-T* 3. They state that the prognosis for lesions of the D.S.S. is graver and that treatment should be more rigid and more prolonged, while infarcts of the D.B.S. are even more serious and may result in sudden death. Consideration of the large proportion of the ventricular musculature which is formed by these two deep muscles (Robb and Robb, 1938) emphasizes the importance which must be attached to an infarct affecting them.

The work of these investigators should receive careful consideration from all those interested in the elucidation of the problems of cardiac infarction.

Rheumatic carditis can cause *R-T* deviation. "Digitalis can produce some depression of the *R-T* segment; but it is invariably negative in all leads, and never of opposite sign in Leads I and III; nor does the *R-T* segment branch from the *R* wave below the iso-electric plane" (Parkinson and Bedford). Changes associated with pericarditis are possibly not quite so well known, but are of great importance, because qualitatively similar to those produced by myocardial infarction. Wood (1937) pointed out that in pericardial lesions there is elevation of the *R-T* segment in all leads, but especially in Lead II in the early stages, and that

[1] The abbreviations used in this quotation refer to the intrinsic muscles of the heart as follows: S.S.S.: superficial sino-spiral; S.B.S.: superficial bulbo-spiral; D.S.S.: deep sino-spiral; D.B.S.: deep bulbo-spiral.

in a later phase inversion of *T* is most marked in this lead. "Pericardial *T* waves" can therefore be distinguished from "coronary *T* waves" by recognition of the *T* 2 pattern and a *T* 2 pattern in association with *T* 1 and *T* 3 coronary waves suggests hæmopericardium. Harvey and Whitehill (1937) reported slightly different findings in tuberculous pericarditis, but commented on persistent inversion of *T* 1 and *T* 2. Application of Robb's work immediately suggests that the superficial sinospiral muscle has been involved by the pericarditis and that the muscle lesion is at least partly responsible for the abnormal electrocardiogram. The electrocardiograms in cases of stab wounds of the heart (Gissane 1937, Wood 1937) provide material for contemplation from the aspects of both muscle damage and pericardial lesions.

Abnormalities of rhythm and of conduction are frequent events in cardiac infarction, and while often recognizable without instrumental aid, may require electrocardiography for their elucidation. Auricular fibrillation and extrasystoles may be indistinguishable if associated with tachycardia, and I have come to view extrasystoles with tachycardia as of definite significance. Paroxysmal tachycardia is serious because of its exhausting effect, and ventricular tachycardia requires urgent treatment. Heart block may occur early or late in varying degree, and always indicates a serious myocardial lesion; variation in the degree of block may be quite rapid.

In connexion with disturbances of conduction, attention must be drawn to the common type of curve in right bundle-branch block, which was described by Evans and Turnbull (1937). The old terminology of bundle-branch block placed right branch lesions as the common variety, but evidence has been accumulating which points to this lesion—tall *R* 1, deep *S* 3, with wide *Q R S* and *T* away from *Q R S*—as being a left bundle-branch block. The hitherto standard curve for right bundle-branch block (new terminology) is rare; but there is a more common type of curve which shows a deep *S* with wide *Q R S* in Lead I and usually in Lead II, *T* upright in Leads I and II and usually inverted in Lead III, *Q R S* usually directed downward in Lead III.

From the foregoing it will be evident that I consider that electrocardiography is essential in the diagnosis and may be of great value in the control of cases of cardiac infarction. It may be the only non-operative means by which this lesion can be diagnosed when an acute abdominal crisis is simulated.

Differential Diagnosis.

The clinical picture of coronary occlusion may be simulated in greater or less degree by many conditions, and it may not be possible to make a dogmatic diagnosis until after the patient has been observed for a period of hours or even days. One of the commonest difficulties is the differentiation from *angina pectoris*; but consideration of the clinical features already enumerated will help materially. Acute abdominal crises are usually accompanied by muscular rigidity, which is infrequent in coronary occlusion; but at an early stage, in the absence of electrocardiographic evidence, acute pancreatitis may be indistinguishable from cardiac infarction, and *vice versa*. Referred pain from a gastric ulcer near the cardia may have a thoracic distribution and be referred to the first and second thoracic segments, resulting in associated pain in the arms. Diabetic acidosis may cause confusion; and acute pulmonary lesions, such as acute infections and spontaneous pneumothorax, may resemble the cardiac condition. The distress of acute pulmonary œdema may accompany an attack of occlusion, as has been mentioned, but as a rule responds readily to morphine and is more commonly due to cardiac failure in hypertension. The complications, such as embolism and pericarditis, may be the presenting manifestations in coronary occlusion; and a search for the cause will reveal it. Pleurisy may be a direct result of recognizable infarction from a mural thrombosis, but when it is not due to infarction evidence of its cause may be found and the type of pain indicates its non-cardiac origin. Chronic myocarditis, so called, should, in the absence of rheumatic carditis and syphilis, be considered to be due to slowly progressive or unrecognized coronary occlusion.

A careful history (often difficult to obtain), close observation, electrocardiography and radiological investigation may all be necessary and may afford the only means of establishing a diagnosis of coronary occlusion.

Prognosis.

Levine (1929) stated that "there is hardly any specific criterion that is decisive or indicative of probable recovery or of the reverse", and it must still be admitted that the prognosis in every case must be extremely uncertain for several days and that no statement regarding even the immediate outlook can be made for about three weeks.

The general immediate mortality in Levine's series was approximately 53%. Strong (1936) compared the figures in 1,275 cases and found that a little less than half the patients were found dead or died within a few hours. Of the patients who survived, about one-half died in the course of a few years, and about one-half of the remainder suffered from congestive cardiac failure or angina, the others being able to resume their occupations (Figure I).

Strong's findings regarding the ultimate result are in keeping with those of Palmer (1937), who recorded the result in 212 cases in which the patients survived the initial attack by three months. More than one-fourth were able to lead fairly active lives, and these lived longer than the remainder, who suffered from angina of effort or dyspnœa on exertion (including chronic congestive failure). The incidence of angina after coronary occlusion was not related to the blood pressure or size of the heart, and the presence of angina did not affect the

average duration of life. The incidence of dyspnœa causing restricted activity followed closely that of the cardiac enlargement, and congestive failure was found only in those with a large heart. The duration of life following the attack averaged 4·2 years, and hypertensives appeared to have a somewhat more favourable outlook than non-hypertensives. Eleven patients (5·2%) were known to have lived for more than ten years. *Angina pectoris* occurred in 39% of cases before the attack, and in 58% of cases following it. "The probability that following recovery from an attack of coronary thrombosis a patient will be able to lead a fairly active life is almost twice as great in those with normal sized hearts as in those with cardiac enlargement."

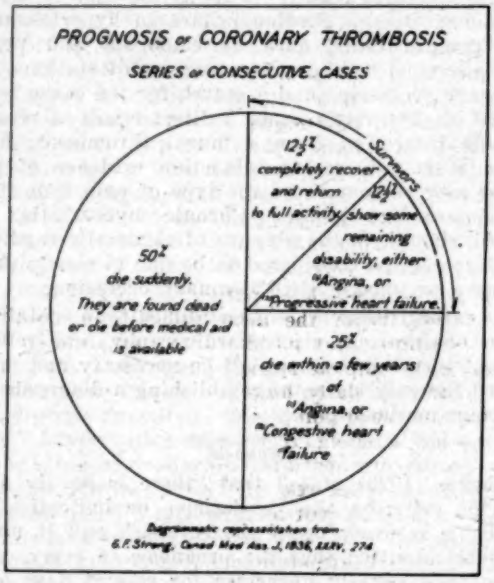
PROGNOSIS OF CORONARY THROMBOSIS
SERIES OF CONSECUTIVE CASES
12½% completely recover and return to full activity
Survivors
12½% show some remaining disability, either "Angina, or "Progressive heart failure.
50% die suddenly. They are found dead or die before medical aid is available
25% die within a few years of "Angina, or "Congestive heart failure
Diagrammatic representation from G.F. Strong, Canad. Med. Ass. J., 1936, XXXV, 274

FIGURE I.

Aneurysm of the ventricle is most frequently due to cardiac infarction (Parkinson *et alii*, 1938), and it occurred in 13 of Wright-Smith's 87 cases of infarction.

The severity of the attack has no relation to the ultimate course, and this is exemplified by recovery from most alarming attacks and by death after apparently mild ones. Death is liable to occur as a result of further infarction, or from some complication directly attributable to the pathological phenomena. Levine was able to study the hearts in twenty-four cases in which the time interval between the onset and death varied from twenty-four hours to several months; twenty patients died within thirty days after the onset of the attack. He found that during the first three or four days the infarcted muscle was deeply red from hæmorrhage. Necrosis was a prominent feature from the beginning, but from the fourth to the twenty-first day was the predominating feature; and this explains the likelihood of rupture during this period. (Rupture occurred in 33% of Wright-Smith's series of 87 infarctions.) Repair was demonstrable as early as the sixth day, but was not a striking feature until after the third week, and more than five weeks were required for cicatrization adequate to prevent possible rupture. Embolism from a thrombus in the ventricle might occur at any time during this period.

The younger patients are a little more likely to recover (Levine, Palmer) and to continue to improve and remain active. Sex does not alter the prognosis, and glycosuria appears to make no difference to the outlook.

Disturbances of rhythm are of importance in that the occurrence of extrasystoles and auricular fibrillation do not appear to affect the mortality, whereas paroxysmal ventricular tachycardia and heart block are of serious prognostic significance. Paroxysmal ventricular tachycardia is amenable to treatment with quinidine, although heroic doses may be necessary; and as demonstrated by the recovery in Case III (to be described later) and by Kerr (1937) "clinical recovery and disappearance of the block is by no means uncommon".

Radioscopy is of undoubted value during the course of the disease as the only really satisfactory means of determining alterations in the size and contour of the heart (Rothstadt, 1938); and kymography is a means of revealing alterations in the muscular contraction (Thomas, 1938,1939).

Treatment.

The treatment of patients suffering from coronary occlusion must be determined by a consideration of the pathology of the condition and must necessarily vary at different stages.

Prophylactic treatment is obviously impossible in those instances in which the first manifestation is the dramatic onset of infarction; but care must be taken when there is evidence indicating coronary sclerosis, such as angina. The keystone of treatment must then be the avoidance of pain and fatigue; and if pain occurs the pain must be relieved as rapidly as possible in order to avoid distress. Vasodilators are undoubtedly of value in relieving the pain of angina of effort, and Evans and Hoyle (1934) have produced convincing evidence that glyceryl trinitrate tablets (trinitrin) are the most effective method of administration, provided that the tablets are dissolved under the tongue. The rapidity of the effect is undoubtedly enhanced by dissolving the tablet in the mouth. I have been quite unconvinced of any beneficial effect from the administration of theobromine or its derivatives; and it should be noted that in most of the proprietary preparations a small amount of sedative is contained in each dose. Sedatives are of value, and my experience has been that just as good an effect is obtained by the use of bromide, phenobarbital or the like. "Cardiazol", given orally, appears to have a miraculous effect in some cases, and to be effective as a prophylactic measure when taken regularly. In several cases the regular taking of "Cardiazol" in the morning and at night has given a better effect than

trinitrin taken similarly and as necessary for the relief of pain; in others it has completely failed to have any effect. Digitalis is rarely of any assistance and often appears to increase the frequency of the attacks. Careful regulation of exercise and rest, the avoidance of gastric distension by the taking of small meals and the use of carminatives, regulation of bowel actions without purgation, moderation with alcohol and rigid restriction of tobacco are all of great importance. A curious feature is that the form of effort may be of importance, and I well remember a patient who could work all day as a stone-mason without ill effect and yet could not walk 100 yards at a slow pace without angina developing.

When congestive failure or a sudden exacerbation of preexisting failure is the presenting feature, the treatment differs only in that the initial symptomatic treatment is unnecessary.

In the classical type of illness the first indication is to relieve pain and distress. The patient must be put to bed in the most comfortable position for him, usually full Fowler's position, and given an adequate dose of morphine or its derivatives. My personal leaning is towards "Omnopon", which is less likely to cause nausea and vomiting, and in the average case at least one-third of a grain of "Omnopon" or one-quarter of a grain of morphine should be given as an initial dose and repeated in half an hour if necessary. In severe cases twice this initial dose should be given, and even that may be inadequate. Repeated doses of the morphine may be necessary and should be given to prevent distress, which will render rest impossible and increase the burden on the myocardium. Counter-irritation is comforting, but heavy poultices can be oppressive.

The myocardium is very intolerant of anoxæmia, and even the slightest degree of cyanosis is an indication for oxygen therapy. Hicks (1939) has devised an easily constructed, simple and light mask which is very well tolerated. The frame is made of light copper wire which is readily bent to the requisite shape, fits on the face like a pair of spectacles and is covered with colourless "Cellophane". By means of it an adequate supply of either oxygen or "Carbogen" can be given. The flow should be at least six to eight litres per minute. When large doses of morphine have been given or when respiration is shallow, 5% "Carbogen" is preferable to oxygen, and it may be given continuously with impunity; but sometimes even these patients are more comfortable with oxygen. If the Hicks mask is not used, administration by means of a nasal catheter is the next best method; but care should be taken to use as large a catheter as can be comfortably inserted. Several openings should be made in the catheter; it must be inserted for a distance of about four inches and lubricated well with a surgical lubricant, such as "Lubafax" or "Uralube"; and the gas must be moistened by being allowed to bubble through water in a Wolffe's bottle. The small oxygen tent can be tolerated for a maximum period of only ten minutes; and a funnel attached to the delivery tube is practically useless as a means of administering oxygen or "Carbogen". Warmth is essential and fresh air is invariably desired. Stimulation is permissible in the first two days and is quite safe; when there has been an excessive fall in blood pressure "Veritol" or "Neosynephrine" is sometimes effective, and "Cardiazol" or "Coramine" appears to be of value and can do no harm.

When collapse is severe the intravenous administration of saline solution may be useful, and hypertonic saline solution has been advocated to aid in the relief of spasm in coronary arteries, a condition which must necessarily occur distal to the site of occlusion, and could inhibit a satisfactory collateral circulation. I have had no experience of its use.

The diet must be small in bulk and therefore is of low caloric value; but in the absence of diabetes it is desirable to give as much sugar as the patient can tolerate. Glucose has no real advantage over cane sugar, but is a little less sweet, and for this reason it is possible to give more of it added to the semi-solid or fluid diet. It is very important to measure the fluid intake and urinary output so that the fluid balance can be determined. It is usually necessary to restrict fluids to thirty ounces per day, so that the tendency to pulmonary and general œdema will be decreased.

The nursing of these patients varies according to the severity of the attack and the degree of cardiac failure; but meddlesome nursing must be avoided. A complete sponging should be given not more than once daily, but great care must be taken with all pressure points. There is no objection to the patient's being recumbent if this does not occasion respiratory distress or cyanosis; in fact he will have better rest and sleep in this position. Purgatives should not be given, but abdominal distension must be avoided. The low residue diet will tend to cause constipation, a desirable state, because less effort will be expended. A bowel action only once a week or even less frequently is not harmful if the patient is comfortable. In many cases it is better to allow the patient to use a commode than to make him submit to a bed pan if he finds difficulty with it.

Apart from morphine and sedatives there is little to be done in the way of medicinal treatment excepting symptomatic treatment. Digitalis is better avoided, because of the risk of ventricular irritability or the degree of heart block, but is necessary if auricular fibrillation occurs. Stimulation must be avoided after the first two or three days, but "Cardiazol" or "Coramine" may be given. Adequate sleep must be assured, and chloral hydrate is perhaps the safest sedative. Small doses of "Luminal" are useful.

"Heparin", which is now available, is a powerful anticoagulant, and experimentally the formation of thrombi can be prevented in animals by its use (Solandt and Best, 1938). As a prophylactic measure it is, of course, inapplicable to man, but its use would probably prevent mural thrombosis

and it might prevent secondary thrombosis in the coronary arteries. Its use is not devoid of risk, but the possible benefit is great; and the reports of its use in thrombosis of the central vein of the retina (Holmin and Ploman, Boström and William-Olsson, 1938), with rapid resorption of the hæmorrhages, suggest that it may decrease the severity of the functional damage of muscle fibres or bundle tissue about the infarct in the myocardium.

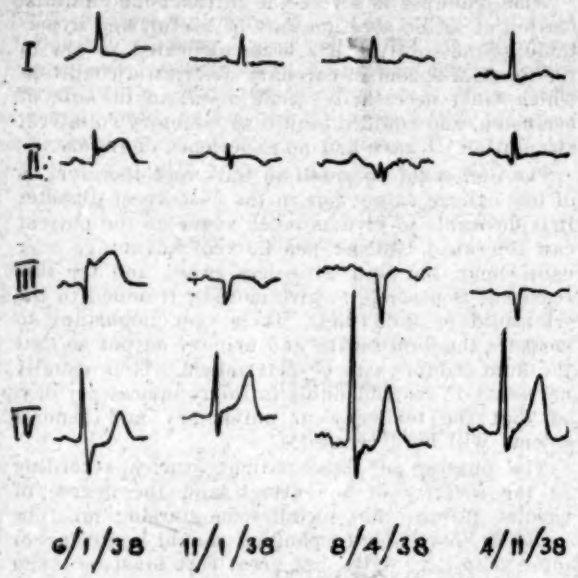

FIGURE II.

Case I. Note *R-T* deviation upward and marked in Leads II and III, downwards and slight in Leads I and IV, and deep *Q* 3 on January 6, 1938. Return of *R-T* to the iso-electric level is evident on January 11, 1938, but there is low voltage in Leads I, II and III, especially in Lead II. *T* 2 and *T* 3 are inverted and of coronary type. On April 8, 1938, low voltage in Leads I and II, W-shaped *Q R S* in Lead II, and coronary *T* waves in Leads II and III are evident. *T* 3 remains inverted on November 4, 1938, although *T* 2 is upright. Voltage has improved. The *P-R* interval was normal throughout.

Irregularities in rhythm call for treatment when they result in increased cardiac activity. Quinidine sulphate should be given, even in massive doses, in ventricular tachycardia, and will often decrease the frequency of extrasystoles; and, in the absence of a rapid ventricular rate and congestive failure, it should be given a thorough trial if auricular fibrillation occurs. Ephedrine may be used in an attempt to prevent the Stokes-Adams syndrome, and barium chloride is said to be of value.

Mercurial diuretics are preferable to other diuretics and should be used if there is a negative fluid balance; one should not wait for the development of manifest œdema. The injections may be repeated every second day, or less frequently if the condition of the patient warrants it. If a satisfactory diuresis is not obtained, ammonium chloride or ammonium nitrate should be given, preferably in gelatine capsules, in doses of fifteen to twenty grains every two to four hours, and continued. "Neptal" and "Salyrgan" are very reliable, relatively non-toxic, and well tolerated. Acute pulmonary œdema and nocturnal dyspnœa are positive indications for the use of mercurial diuretics. It may be necessary to give injections once or twice a week for many weeks after convalescence has been established. As a result of the use of these diuretics, *paracentesis thoracis* is necessary much less frequently than it used to be. The duration of the period of rest in bed may be determined by the presence of signs and symptoms of cardiac failure; but from consideration of the pathology it is evident that even in the presence of good cardiac efficiency it is necessary for the patient to avoid exertion for six to eight weeks, that is, until there has been time for firm healing to occur. Despite apparently good cardiac efficiency, it is unjustifiable to put the patient to the test if there is clinical or electrocardiographic evidence of coronary occlusion. In Case II the patient, a male, who gave a typical history, was made to run upstairs twenty-four days after the attack to test his tolerance, and was told to resume his work as traveller. Needless to say, I refused to permit it.

Convalescence must be prolonged and a gradual return to exercise is permitted only when there is no evidence of cardiac failure.

Associated conditions are sometimes of great importance in treatment, and of these *diabetes mellitus* is perhaps the most important. There

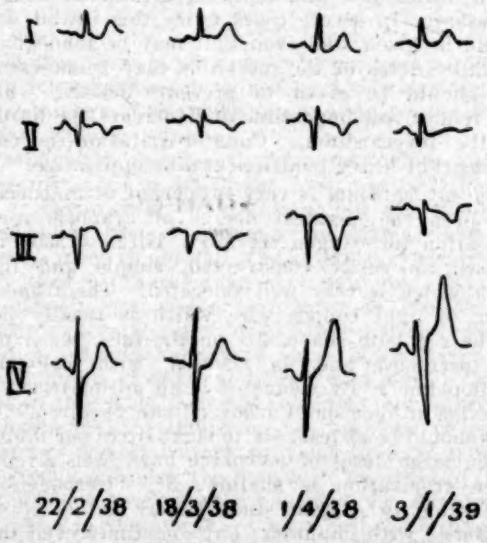

FIGURE III.

Case II. Tracing on February 22, 1938, ten days after the attack of occlusion, shows *R-T* deviation with elevation and coronary *T* waves in Leads II and III. *Q* 2 is deep. Coronary *T* waves persist in Leads II and III on March 18, 1938, and April 1, 1938; there is no *R-T* deviation; *Q* 2 is still deep. Although *T* 2 is upright on January 3, 1939, *Q* 2 remains deep and the coronary *T* wave is still evident in Lead III, eleven months after the attack.

should be no hesitation in the use of insulin if the glycosuria is not readily controlled; but it is better to allow a little glycosuria than to risk hypoglycæmia. Protamine zinc insulin has some advantages because of the use of frequent small

feedings. Other associated diseases must be treated as necessary, the cardiac disease being borne in mind.

The question of surgery after cardiac infarction is one which is increasing in importance, but I have had no experience with it as yet. O'Shaughnessy (1937) described a form of operative procedure and the indications for operation have been pointed out by Davies and others (1938). The indications may briefly be described as symptoms and signs of cardiac ischæmia, due to coronary disease or syphilis, in a patient under about sixty-five years of age, in whom there is either a disease of progressively increasing severity, or a greater risk from conservative than there is from an operative procedure. To me cardiac aneurysm is not an indication for surgery.

Illustrative Case Reports.

CASE I.—A.W., a male, aged seventy-five years, was seen on July 23, 1937, and gave a history that indicated an attack of coronary occlusion twelve days previously. There was no clinical evidence to be found and the electrocardiogram showed only a low voltage. He made a complete recovery.

On January 6, 1938, a typical attack of occlusion occurred and a tracing taken one and a half hours after the onset showed *R-T* deviation with a high take-off in Leads II and III.

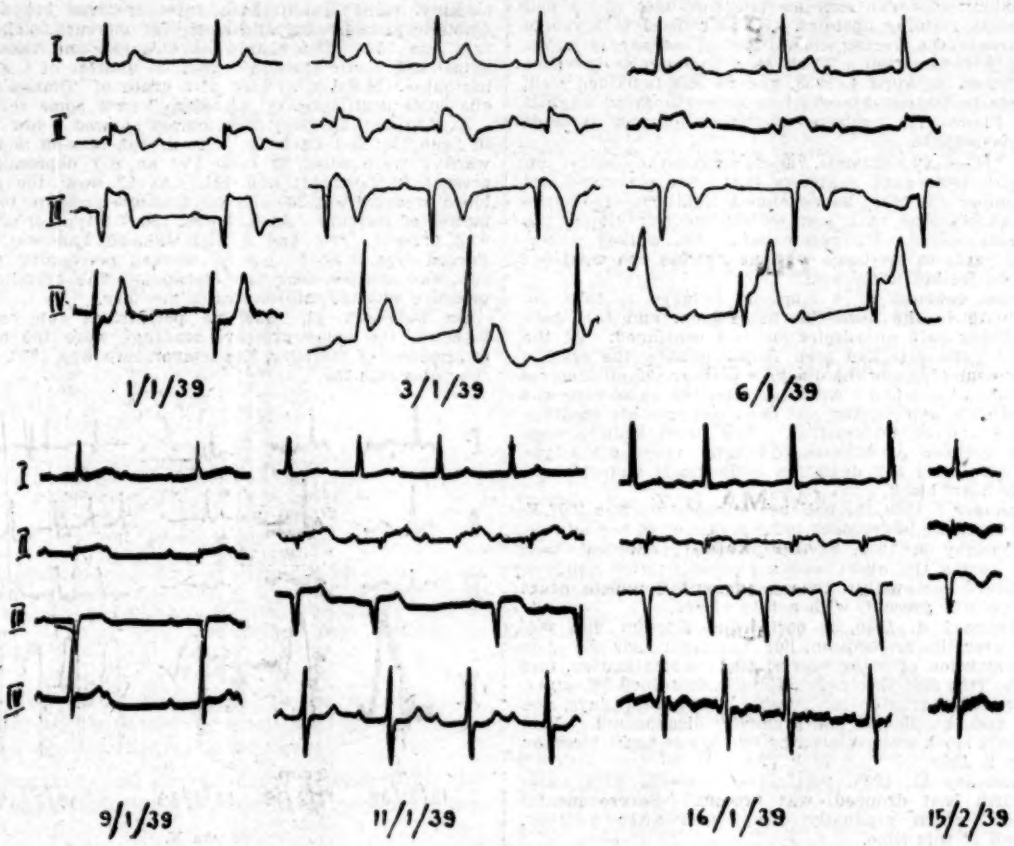

FIGURE IV.

Case III. On January 1, 1939, approximately six hours after the onset of severe pain and twenty hours after the first symptoms, complete heart block with a ventricular rate of 52 per minute is evident. *R-T* deviation is marked in Leads II and III, and slight in Leads I and IV F. *T* 2 and *T* 3 are inverted. The ventricular rate, 65 per minute on January 2, 1939, was 60 on January 3, 1939, still with complete heart block. There is less marked *R-T* deviation. *T* waves are of coronary type in Leads II and III. On January 6, 1939, complete heart block is still evident; coronary *T* 3. On January 9, 1939, 2:1 heart block is evident; coronary *T* waves not seen. Voltage has become progressively lower in Lead II. Partial heart block is evident on January 11, 1939, about every fifth beat being dropped, and the *P-R* interval gradually increases in length prior to the dropped beat. Normal rhythm, with a *P-R* interval of 0·2 second preceded auricular fibrillation shown in the tracing on January 16, 1939. Coronary *T* 2 and *T* 3, low voltage in Lead II, and normal rhythm are evident in the tracing on February 15, 1939.

On January 11, 1938, coronary *T* waves were present in Leads II and III.

On January 19, 1938, another typical attack occurred some hours after the tracing had been taken, and the nurse reported that the pulse rate was very slow—only half the previous rate; this was presumably due to a temporary 2:1 heart block.

The patient's subsequent course was uneventful and he has remained well; but inversion of *T* 3 is still present. Electrocardiograms are shown in Figure II.

CASE II.—W.G., a male, aged thirty-nine years, reported on February 22, 1938, a typical attack of severe pain which had occurred on February 12, 1938, and which had lasted for six hours. The pulse rate was said to have been 48 per minute. No abnormality was found on physical examination; the blood pressure was as had been found on previous examinations—140 millimetres of mercury systolic and 84 diastolic. An electrocardiogram showed deviation of *R-T* in Leads II and III, with a high take-off and coronary *T* waves.

He was sent home to rest in bed and reported on March 18, 1938, that ten days previously (twenty-four days after the attack) he had been told to resume work after being submitted to an exercise tolerance test which had consisted in running upstairs. I had refused to agree to this course, and a tracing showed typical coronary *T* waves with no *R-T* deviation. Work as a commercial traveller was resumed on April 4, 1938, and he has remained well, but needs to restrict his effort in order to avoid anginal pain. Figure III contains electrocardiograph tracings from this patient.

CASE III.—G.C., a male, aged sixty-seven years, on January 1, 1939, gave a history that after playing bowls on December 31, 1938, he developed tightness across the chest and stabbing pain just within the left nipple, the pain being worse with respiration. He walked a few hundred yards to his home without distress and went out that night feeling fairly well.

He was wakened at 5 a.m. on January 1, 1939, by aching pain in the lower sternal region, and this pain and stabbing pain on respiration had continued. At the onset his pulse rate had been 80 per minute, the systolic blood pressure 140 and the diastolic pressure 90 millimetres of mercury; but when I examined him the pulse rate was 52 per minute, and regular, and the blood pressure readings were 136 and 90 millimetres. The heart sounds were scarcely audible. An electrocardiogram showed a *T* 3 type of occlusion with *R-T* deviation in Leads II and III, and complete heart block.

On January 2, 1939, the patient's temperature was 100° F. He had complete heart block, with a rate of 65 per minute.

On January 3, 1939, severe pleuritic pain had been present during the night, and a pleural friction rub was easily heard just within the apex beat. Complete heart block was still present, with a rate of 60.

On January 4, 1939, a pericardial friction rub was audible over the precordium, but maximum near the apex beat. Retention of urine necessitated catheterization, and the urine (the first specimen obtained) contained 3% sugar. The pericardial friction rub persisted for several days, and both it and the pleural rub gradually disappeared. Complete heart block was replaced by two to one heart block on January 9, 1939.

On January 11, 1939, partial heart block, with about every fifth beat dropped, was present. Severe mental confusion, which gradually disappeared after a week, developed at this time.

On January 12, 1939, normal rhythm returned. The blood sugar content at 11 a.m. was 0·39%. Treatment with insulin was instituted.

On January 14, 1939, occasional irregularity of rhythm was present. A negative fluid balance and signs of effusion at the left base (confirmed radiologically) were indications for the use of diuretics, and "Neptal", one cubic centimetre, was given intramuscularly with good effect and thereafter continued twice weekly.

On January 15, 1939, auricular fibrillation was evident and confirmed with a tracing on the following day.

On January 17, 1939, normal rhythm, with a rate of 76 and a *P-R* interval of 0·2 second, was present.

On January 19, 1939, quinidine sulphate was given because of recurring paroxysms of auricular fibrillation. A controlled diet was now possible, and the amount of insulin was gradually reduced, until he was able to leave hospital early in March after an uneventful course during the later stages. With improvement in the general condition there appeared to be an improvement in the carbohydrate tolerance, and it was possible to stop the administration of insulin.

He has gradually resumed his activity, although retired from regular work, and has remained well. A tracing on June 30, 1939, showed coronary *T* waves in Leads II and III. Earlier tracings, which illustrate the variations in *R-T* deviation, in *T* waves, and in rhythm, are shown in Figure IV.

CASE IV.—W.S., a female, aged seventy-five years, was seen at 6 a.m. on February 12, 1939, because of the sudden onset of pain across the chest, a sense of compression and repeated vomiting, commencing at 3 a.m. The pain had persisted. There was a past history of arterial hypertension, and she had been subject to a sense of oppression over the sternum on exertion. She was shocked, cold and clammy. The systolic blood pressure was 210 and the diastolic pressure 106 millimetres of mercury. The pulse rate was 106. The heart was enlarged and there were apical and aortic systolic bruits. A quarter of a grain of morphine (M.S.A.), a third of a grain of "Omnopon" and one cubic centimetre of "Cardiazol" gave some relief.

At 7.30 a.m. an electrocardiogram showed a low voltage in Lead III; *R-T* deviation with a high take-off and downward *T* were noted in Lead IV; an *R-T* depression was present in Leads II and III. At 12 noon the systolic blood pressure was 200 and the diastolic pressure 106 millimetres of mercury. At 5.30 p.m. the *T* 1 type of occlusion was present. *T* 4 had a high take-off and was of the Pardee type. At 10 p.m. a marked pericardial friction rub was audible near the sternum. The systolic blood pressure was 190 millimetres of mercury.

On February 13, 1939, no pericardial rub could be detected, the blood pressure readings were 160 and 100 millimetres of mercury, the temperature was 100° F., and the pulse rate 110.

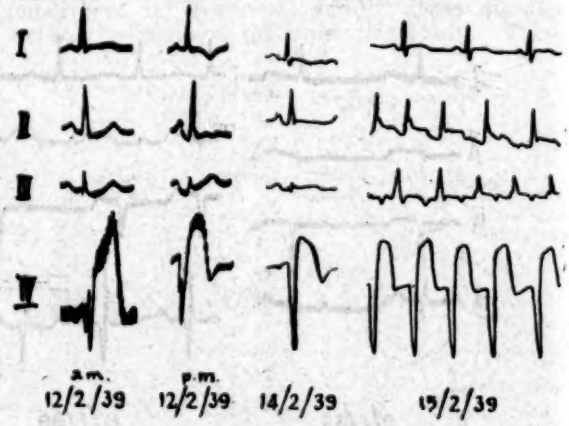

FIGURE V.

Case IV. The morning tracing, taken four and a half hours after the onset of pain, shows gross *R-T* deviation in Lead IV F, slight deviation in Leads II and III, and low voltage in Lead III. The evening tracing (ten hours later) shows the anterior type of occlusion with a coronary *T* wave in Lead I. *T* IV F is inverted and there is great elevation of the take-off in this lead. Further decrease in voltage is evident in Lead III. On February 14, 1939, the voltage is very low in Lead III; *T* 1 is only slightly inverted, *T* 2 is iso-electric; *P* 4 and *T* 4 are inverted. Tachycardia with extrasystoles of supraventricular origin is evident in Leads II, III and IV on February 15, 1939. *T* 1 is already upright, but there is upward displacement of *R-T* in Lead II, *T* 3 is inverted and Lead IV is grossly abnormal. Although the patient was much worse, the voltage is normal in Lead III. Lead II suggests the occurrence of hæmopericardium.

On February 14, 1939, the systolic blood pressure was 146 and the diastolic pressure was 80 millimetres of mercury, the temperature was 99·4° F., and the pulse rate was 108. There was severe pain in the tip of the left shoulder and over the upper part of the thorax on the left side; the pain was worse with respiration. A slight change in the tracing was evident.

On February 15, 1939, paroxysms of tachycardia and frequent extrasystoles were present with an increased heart

rate. There was no response to quinidine, and the general condition steadily deteriorated, to result in death, in coma, on February 16, 1939.

The alteration in the types of curve between February 14 and 15, 1939, suggests the possibility of hæmopericardium having developed (Figure V).

CASE V.—B.W., a male, aged fifty years, on September 24, 1938, complained of typical anginal pain of three months' duration, gradually becoming worse, and at the time occurring after a walk of two hundred yards at an average pace. It was also produced by excitement or stress.

The blood pressure readings were 156 and 100 millimetres of mercury; the pulse was irregular and varied in rate, without apparent reason, from 72 to 90 per minute; the irregularity was unaffected by deep respiration. General examination revealed no definite abnormality.

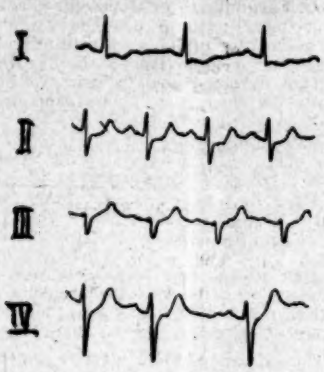

FIGURE VI.

Case V. Angina of effort. In addition to left ventricular preponderance there is definite deviation of *R-T* in Leads I, II and IV F. The irregularity in rhythm is physiological.[1]

An electrocardiogram showed a low take-off of *R-T* in all leads, but especially in Leads I, II and IV F, and slight left ventricular preponderance. The irregularity was of vagal type (Figure VI). The patient died suddenly in February, 1939.

Summary.

1. A brief reference is made to the pathology in disease of the coronary arteries and the effects of ischæmia on the cardiac musculature.

2. Clinical types of coronary disease and the clinical manifestations of coronary occlusion are detailed.

3. Electrocardiographic findings after cardiac infarction are described, and the work of Robb and Robb regarding muscle bundle localization of infarction is brought forward. The importance of changes due to pericardial lesions is discussed, and attention is directed to the common type of right bundle branch block described by Evans and Turnbull.

4. The differential diagnosis and prognosis are discussed.

5. The treatment of patients suffering from coronary occlusion is described in detail.

6. Five illustrative cases, with serial electrocardiograms, are reported.

[1] The tracings in this article were taken with the Both electrocardiograph, and in their preparation for publication time markings have not been reproduced.

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Reviews.

SEX IN EVERYDAY LIFE.

In his introduction to "Sex in Everyday Life"[1] Sir Walter Langdon-Brown says:

> The thinking public is clamouring for reliable information on matters which so deeply concern human health and happiness. Here they will obtain it; myths, misstatements and superstitions are swept out of the way, and wise, sympathetic guidance is provided.

Dr. Griffith's wide knowledge and culture enable him to discuss not only the scientific but the religious and ethical sides of his subject with authority. His references to sex and religion in primitive peoples are interesting as an illustration of the part played by the laws and ritual of sex relationships in the history of civilization. He points out that:

> Far from living lives of "sexual license" many primitive societies possessed rigid and complicated rules with regard to sexual behaviour.

While his aim is the spread of scientific knowledge, he would wish to preserve something of this age-old reverence for the natural urges and instincts. He emphasizes the point that science alone cannot give a complete guide to conduct, "for the simple reason that science does not provide us with a method of life, it only presents us with facts and truths".

The style of the book is attractive; but the arrangement of the matter might perhaps be more happy. To begin a book intended for intelligent lay readers with unattractive anatomical diagrams and descriptions seems a little drastic. The tendency to reduce everything to catalogues and tables, which is the curse of our scientific education, takes an almost ludicrous form at times—for instance, when women are solemnly divided into four groups according to their attractiveness. Those of group I are irresistible—sweep the man off his feet; groups II and III are mercifully less and less devastating; while those in group IV, unhappy wretches, are entirely lacking in any attractive qualities at all.

With its vast mass of information and statistics, its careful bibliography and its interesting presentation, this book will be of great value to both medical and lay people whose work and interests involve some understanding of this important subject.

[1] "Sex in Everyday Life", by E. F. Griffith, M.R.C.S., L.R.C.P., with an introduction by Sir Walter Langdon-Brown, M.D., D.Sc., F.R.C.P., and a foreword by H. A. Gray, D.D.; 1938. London: George Allen and Unwin Limited. Demy 8vo, pp. 378. Price: 10s. 6d. net.

MEDICAL SOCIETIES.

"CERTAIN gentlemen desirous of improvement in medicine and other sciences nearly allied to it, and convinced of the great advantages arising from a free communication of observations and opinions, have determined for that purpose to enter into an association which they chuse to distinguish by the name of the Physical Society. And perceiving how much the attainment of the advantages which they expect from this institution may be facilitated by proper Rules, they have agreed to submit to the following."

Could the proper functions of a medical society be more admirably expressed than in this passage from the preamble to the original book of laws of the Guy's Hospital Physical Society? The society was founded in 1771, and in its heyday received many communications still of great historical interest. At a meeting in 1795 "Mr. Davis related that two bodies were brought into the dissecting room at the same time. On the second day one had become putrid and was covered with maggots. The other next to it was avoided by flies; this was the body of a man who had been heavily dosed with mercury for syphilis. Mr. Davis asked whether this was the reason why the flies had left the body alone"—a hint of the antiseptic properties of mercury. The Physical Society is one of more than thirty British medical societies whose stories have appeared in *The Medical Press and Circular* during the past few years and are now collected in a single volume.[1] The history of British medical societies is in a way the history of British medicine and of medical practice and practitioners in all parts of the country. In this book we meet many famous men, and others who, although less well known, loved to bring their fellows together to learn and teach, to discuss their common problems, or just to eat and drink and be merry; these are the founders and the resurrectors of societies. This entertaining history book will bring a wider perspective to the medical man who has the welfare of some society near to his heart and has pored over the records of its ups and downs.

[1] "British Medical Societies", edited by D'Arcy Power, K.B.E., F.R.C.S.; 1939. London: Baillière, Tindall and Cox, for The Medical Press and Circular. Demy 8vo, pp. 328, with 48 plates. Price: 10s. 6d. net.

A MANUAL ON MARRIAGE.

AN Australian edition of Hannah and Abraham Stone's "Marriage Manual" will be welcomed because of the simple and practical way in which it answers the questions which most people must ask some time in their lives.[2]

We may be inclined to underestimate the need for such books nowadays, when apparently the young person is a good deal better informed about the "facts of life" than the parent who, like the embarrassed father in Somerset Maugham's play "The Breadwinner", thinks it his duty to give his offspring instruction on the subject. It is hard to realize what unhappiness and tragedy taboos and ignorance of sex questions caused in the past. That a J. Kay Chapman should have had to rush home after a quarrel, hold his hand over a fire until it was burnt to the bone, and suffer weeks of pain before he could acknowledge to himself that the feeling he had for the beautiful Italian girl who read Dante with him was really love, is almost incomprehensible to our generation.

It is the correction of half-knowledge and cynicism as well as ignorance and false modesty that is the aim of such books as "Marriage Manual", and their popularity is due not to a more frivolous but a more serious and reverent attitude to sex problems. The book is readable because of the short and succinct answers to questions which occur to everybody, although the questioners, as young lovers, lose something of their romantic glamour in the atmosphere of the clinic and the consulting room. The claim of the editor that it is "the best Baedeker to matrimony that has ever been published" is not difficult to sustain. The vast clinical knowledge of the authors, enabling them continually to quote actual cases, gives them a great advantage over mere theoreticians and always carries assurance and conviction.

We would agree with the editor that, though "knowledge is not a complete safeguard, ignorance is none the less a grave danger", and that it would be hard to find a book in which "this indispensable knowledge is imparted so precisely or so decently as in *A Marriage Manual*".

[2] "A Marriage Manual: A Practical Guide Book to Sex and Marriage", by H. M. Stone, M.D., and A. Stone, M.D.; 1939. London: Victor Gollancz Limited, in association with John Lane; Australia: Angus and Robertson Limited. Large crown 8vo, pp. 325. Price: 12s. net.

The Medical Journal of Australia

SATURDAY, SEPTEMBER 30, 1939.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE MEETING OF THE FEDERAL COUNCIL.

MATTERS of considerable moment occupied the attention of the Federal Council of the British Medical Association in Australia at its recent meeting, which is reported in this issue. With the exception of Tasmania, whose representatives were unable to attend on account of the state of national emergency, all the States were represented; and the deliberations were, more than ever perhaps, marked by sincerity and a will to work for the good of the profession and the public. The outbreak of war a few days prior to the meeting doubtless had something to do with the "atmosphere" of the gathering, and it was noticeable that the council held in abeyance all items on the agenda paper which had become relatively insignificant or inopportune in view of the national crisis.

Finance required a great deal of consideration, for it was obvious that the Federal Council would either have to raise money to enable it to carry on its activities or else to curtail them. If the activities of the council were to be extended, it was clear that it would need more money than it had previously possessed. From the report of the meeting readers will see that the council had before it a letter from the Parent Body setting out certain proposals which had been formulated for the benefit of the South African Branch of the Association. These proposals show that the Parent Body is doing what it can to help the Australian Branches and to satisfy their needs as to both finance and autonomy. It was pointed out at the meeting that though the South African Branch and the Federal Council in Australia have a somewhat similar financial problem, the environment and the working conditions in the two countries are essentially different; and that what may provide a solution for the difficulties in one set of circumstances will not necessarily be successful in another. In due course these proposals will be brought before the members of the several Branches for their consideration, and it is to be hoped that those who bring the matter before the members will be thoroughly conversant with all the aspects of the question. In these circumstances it is not the province of this journal to express an opinion either in favour of or against the proposals. We may, however, state, and indeed we should fail in duty were we not to state, that at the present critical time in the nation's history no step must be taken which would suggest a possible weakening of the link which binds us to the Home Land. It is for members to determine whether this close bond of union would be weakened or perhaps jeopardized if the proposed arrangement were accepted and every member of the Association in Australia did not receive *The British Medical Journal* each week from England, as he does at present.

It was inevitable that the date of the sixth session of the Australasian Medical Congress (British Medical Association), which was to have been held in Perth, Western Australia, next September, should be cancelled. Members are asked to note that, though the date is cancelled, the work of the executive committee has merely been suspended. Unfortunately this is the second time that the medical profession in Perth has been disappointed in the holding of a session of congress. A great deal of useful work had already been done,

and the executive committee had received much encouragement from Branches in other States and from medical organizations outside the Association. This effort will not be wasted, for all that has been gained by it will serve as a basis for future action.

In last week's issue reference was made to the resolution adopted by the council in regard to its cooperation with the Federal Government in the defence of Australia. Members of the Association will naturally wish to know whether any steps are being taken to safeguard the practices of medical practitioners who may be called up or volunteer for active service either at home or abroad. A scheme was drawn up and approved by the Federal Council and will be submitted to the Branches for the consideration of their members.

The South Australian Branch has done a service to the community by its insistence on the provision of an adequate pension for tuberculous patients and their dependants. It is unnecessary to reproduce here any details of the discussion or the motion which was carried; but it may perhaps be pointed out that even if the sum paid in an invalid pension were increased, this would not meet the case. The invalid pension is for persons who are permanently and totally incapacitated. A patient in the early stages of tuberculosis is by no means permanently incapacitated from earning his living. If he is given proper treatment he has every hope of recovery and of being once more a useful member of society. The institution of a family endowment on the lines suggested in the Federal Council's resolution would be a far-sighted and useful form of investment or insurance for national health. What is needed is a statesman with vision who will take up this matter and insist on seeing it through.

One last point may perhaps be mentioned. The members of the Federal Council have to deal with subjects very similar to those considered by Branch councils. In fact most of the items on the agenda paper come to the Federal Council from the Branches. Zeal and even breadth of vision are not alone sufficient to make a good Federal councillor. A councillor must have the confidence of his Branch council and should have had experience of Branch council work. For this reason we are glad that no alteration was made in the method of election of Federal councillors by the Branch councils. To be a member of the Federal Council is no sinecure; it demands both time and self-sacrifice. We think that most Branch members realize this and appreciate the work of the Federal Council.

Current Comment.

HEPARIN AND THROMBOSIS.

ANTICOAGULANTS have been used for many years now for preventing the coagulation of blood *in vitro*. Potassium oxalate, sodium citrate and half-saturated sodium sulphate are of this class. Only recently, however, have organic substances of animal origin been used *in vivo* to prevent intravascular clotting. Of these, heparin has now received an extensive experimental and clinical trial, and bids fair to having a wide usefulness in medicine and surgery. Most of the experimental work on heparin has been carried out in the department of physiology in the University of Toronto, under the guidance of Professor C. H. Best.

Heparin (ἧπαρ, the liver) was first separated by McLean in 1916 in Howell's laboratory, from the liver tissue of the dog. The yield from this source was very small, but was sufficient to prove its role in the coagulating of blood as an antiprothrombin and also, to a lesser extent, an antithrombin. A more fruitful source of heparin was demonstrated in the organs of the ox, and the Canadian heparin is now derived from beef lung, some hundred pounds of which yield one gramme of heparin. It has now been purified by Charles and Scott as a crystalline salt of barium of uniform potency. It has also been standardized, and the blood heparin can be assayed by its combining power with protamine. Heparin first entered the field of experimental surgery in 1932, when Gordon Murray, of Toronto, used it to prevent the formation of thrombi in blood vessel anastomoses. It proved itself eminently successful if the animals were given heparin for from seven to ten days prior to anastomosis. Heparin appears to be produced in the mast cells of Ehrlich, which occur in relation to various blood vessels. It is produced in very high concentration in the dog in anaphylactic and peptone shock in such large quantities as to render the blood practically incoagulable. Clinical trial now extends to some 400 cases, including diseases of various kinds, in none of which has any untoward or toxic effect been produced as a result of the administration of heparin.

Apart from cardio-vascular surgery, peripheral embolectomy *et cetera*, in which the drug found its first and possibly greatest usefulness, it has received a fair trial in the prophylaxis of post-operative pul-

monary embolism. In one series of cases administration of heparin was commenced from four to twenty-four hours following a major surgical procedure of the type prone to be followed by pulmonary embolism—partial gastrectomy and large gynæcological operations. The incidence of post-operative pulmonary embolism in this class of operation was some 2% to 7% in a control series. Amongst the patients who had been treated with heparin there was no mortality from this cause. Administration of the drug was not commenced till some four hours after the operation, so that it could not be blamed for hæmatomata or bleeding from the operation site. In point of fact, these complications did not show a greater incidence in the patients treated with heparin than in the control series. Sufficient heparin was given to ensure that the coagulation time of the blood was doubled or trebled, and was continued for about ten days after operation. In cases of established thrombosis in a vessel the effect of heparin appears to be in the direction of preventing the extension of further white clots. It is not suggested that the concentration of the drug which can be safely attained in the human subject will in any way "dissolve" the thrombus—an irreversible change. Heparin can also be used in the prevention of coagulation in the process of blood transfusion. One milligramme (100 units) of heparin per kilogram of body weight is administered to the donor and the blood is withdrawn ten minutes later. At the end of two hours the coagulation time of the donor returns to normal.

Apart from the use of heparin in the prevention of thrombosis in the systemic circulation, its effect in the prevention of clotting in the portal circulation has recently been investigated by G. Murray and R. McKenzie in the department of surgery at the Toronto General Hospital.[1] Experimental surgery on the dog first established the safety of the use of heparin, and its clinical trial was then applied to the prevention of thrombosis in cases of splenectomy and in cases of mesenteric thrombosis following ablation of the thrombosed segment of gut, both of which conditions are notorious for the frequency of a spreading thrombosis in the portal system following operation. In the eight cases of splenectomy reported, followed by heparin therapy, there was no portal or systemic thrombosis. Six patients suffering from mesenteric thrombosis, in which the affected segment was resected, showed no further thrombosis or gangrene, although two of them died from other causes. Murray and McKenzie are careful not to draw general conclusions from such a small series of cases. They do state, however, that when many surgeons have not seen recovery occur in a case of mesenteric thrombosis associated with gangrene, their results are probably significant.

In conclusion it may be stated that heparin is not yet being prepared in large enough quantities for extensive clinical trial, and that the price at present is prohibitive. A Swedish preparation which is used in England and elsewhere contains a fairly high percentage of impurities and has produced toxic effects. As soon as an inexpensive synthetic product is on the market, heparin should have further clinical trial. It may, and possibly will, find a wide field of usefulness in the spheres indicated above.

[1] *The Canadian Medical Association Journal*, July, 1939.

THE DIFFERENTIAL LEUCOCYTE COUNT.

A MOMENT'S thought should be sufficient to convince one that there are sources of possible fallacy in the differential leucocyte count as usually conducted. In the ordinary blood film leucocytes congregate in large numbers at either end, and they are much more numerous at the edges than in the middle. It has also been suggested that it is the granular cells and monocytes especially which tend to move to the edge of the film. The count will be incorrect if the edges only, or the middle portions only, or the area close to one end or the other, are examined. Most workers assume that if they count a large number of cells, examining edges as well as central areas, the result will be accurate enough. R. M. Boveri, however, declares that this method produces fallacious results, no matter how careful the observer may be.[1] In order to make an accurate estimate of the relative proportions of lymphocytes and other cells, he counted the leucocytes in blood, diluted ten times with Thoma's fluid, in a Buerker chamber. He states that: "Although it is difficult in the Buerker chamber to distinguish between monocytes and the various types of granulocytes, it is very easy to recognise the lymphocytes by their size, nucleus and cytoplasm." He compared the results obtained by this method with those obtained from the examination of ordinary blood films and films made on cover glasses. A film is made on a cover glass in the following manner. A drop of blood is placed on a cover glass and another cover glass is placed on top of it; the blood spreads out between the two glasses, which are then separated by a sliding motion. This method also fails to effect an even distribution of leucocytes.

When one examines a blood film made by the second method, on cover glasses, one again finds that the distribution of the white cells is not an entirely random one. The white cells tend to congregate more thickly towards the centre of the cover glass; and since this tendency is again more marked among the granulocytes and monocytes, the lymphocytes are relatively more numerous towards the periphery of the cover glass. This departure from a perfectly random distribution is not very marked; in fact the effect is so slight that a differential count made by this method may reasonably be expected to give a result very close to the truth.

Boveri made a large number of counts by each of the three methods, counting large numbers of cells on each occasion. The results obtained by

[1] *Guy's Hospital Reports*, January, 1939.

the Buerker method corresponded closely with those obtained by the cover glass method; but the lymphocyte percentage obtained by the method ordinarily employed was on an average higher by about ten. He finds that the true normal lymphocyte percentage ranges from 20 to 30 and not, as is generally believed, from 25 to 40.

In the interests of accuracy, confirmation of Boveri's results by some other worker would be of importance. Whether the knowledge will prove of value in other ways is another matter. Most people will continue to use the methods they use now. It probably does not matter which is used, provided it is understood that different methods give different results.

It is a pity that Boveri did not confine his observations to his counting and the comparison of his counts. The person who is not a mathematician will perhaps accept the following statement without having to worry over it; a mathematician will not accept it at all.

> . . . the slide method gives a numerical excess in the lymphocyte percentage of about ten points. A simple calculation shows that this must mean that, in relation to the white cells, about four-tenths of the original number of granulocytes and monocytes are removed from the countable part of the film.

In our opinion, such a calculation, far from being simple, is impossible. The proportion of leucocytes at the ends of the film (that is, where the cells cannot be counted) will depend on the size of the drop of blood, the pressure exerted, the length of the film in proportion to the size of the drop, the width of the film, and various other factors.

THE TREATMENT OF SEVERE HÆMORRHAGE DUE TO PEPTIC ULCER.

IT is somewhat surprising to read in a recent article by John La Due, on the treatment of massive hæmorrhage due to peptic ulcer, that clinicians cannot agree on the question of giving or withholding food from patients who have suffered this variety of blood loss.[1] It is also surprising to be told that most clinicians advocate a period of fasting in these circumstances, because, judging by what is heard locally in discussions on the treatment of hæmatemesis and melena, the teachings of the Meulengracht school have been readily accepted in this country. But as it is impossible to take a census of medical practice, and as it is apparent that there are still those who adhere to the older methods, it is worth giving a little attention to the series of 82 cases described by this author, and this in spite of a recent discussion on the subject in these pages on August 19 last. Of 79 patients under controlled conditions, five died as the result of repeated bleeding from peptic ulcer, a mortality of 6·3%; but when the deaths are carefully analysed it appears that the true mortality rate was much lower when the advised technique of treatment was followed, being only 1·3% (if it is justifiable to refer to percentages in so small a series). Again and again it is to be seen in the history of medicine that little heed is taken of doctrines or essential details of techniques when they are first described. We have all heard of the Lenhartz diet; but how many of us know that Lenhartz in 1906 introduced the important principle of feeding patients immediately after the occurrence of hæmorrhage from a peptic ulcer? La Due reminds us that this is so, and that the mortality rate in the series of 146 patients so treated by Lenhartz was only 2·14%, a figure which comes within reach of the 1% with which Meulengracht threw his challenge to the reactionaries. The recommendations of this latter worker in Copenhagen should be now well known to all; perhaps it was, and perhaps it still is, a shock to some physicians to contemplate feeding these patients liberally with all soft foods without placing special restrictions on appetite; but it is just this method of keeping the stomach partly filled which has given such excellent results.

La Due follows the routine advised by Andresen in 1916, which differs from the above in some respects, but is based on the same general principles. In the Andresen diet gelatin is employed during the first few days; it does not reach the level employed by Meulengracht for a week or more, but the stomach is not allowed to remain empty and the patient is kept well nourished by an adequate ration. The general routine followed in La Due's series is as follows. The patient is kept quiet for the first day by morphine or by bromides with chloral hydrate, and is placed on the advised fluid and semi-fluid diet at once. A blood count is made and the blood is typed, though no transfusion is given as a routine during the first week unless air hunger or considerable fall in blood pressure is observed. It will be seen that this differs considerably from the practice followed by Witts and others in England and Australia; but perhaps no standard can be laid down that will supplant the clinical judgement of the medical attendant. The main thing to remember is that anæmia and anoxæmia may be highly dangerous; if such danger threatens, a transfusion should be given so as to avert changes in essential organs, such as the brain and kidneys, changes that may damage them beyond repair. La Due advises specifically that certain things should not be done: that ice should not be applied externally or given by mouth, and no food or drugs given other than those ordered; that the blood pressure should not be suddenly raised by the parenteral administration of fluid; and that adrenaline and other pressor drugs should be avoided. The Andresen treatment is planned to meet the physiological requirements of the patient without going so far as the more revolutionary Meulengracht régime, and provides for frequent small feedings, which incorporate a bland gelatin mixture designed to combine with the gastric secretions. The results have apparently been very

[1] *Archives of Internal Medicine*, June, 1939.

encouraging and should help to convince any who may still doubt the value of early feeding after hæmatemesis and melena.

Attention might perhaps be directed to the question as to whether frequent feedings are in any sense truly physiologically sound. A routine which approximates more closely to the normal digestive habits of the individual seems to be preferable, and there are not wanting critics who maintain, and probably with good reason, that all systems of the treatment of peptic ulcer based on this method of frequent divided feedings are based upon a fallacy. However, this is perhaps a matter of detail. The point is that with early and adequate feeding the patient who has suffered a recent bleeding from a peptic ulcer is more comfortable, recovers more quickly and is faced with decidedly less peril.

THE WALTER AND ELIZA HALL INSTITUTE OF RESEARCH IN PATHOLOGY AND MEDICINE.

THE Walter and Eliza Hall Institute of Research in Pathology and Medicine was founded in 1916. The trustees of the late Walter and Eliza Hall completed the pathological block of the Royal Melbourne Hospital and provided a sum of £2,500 annually towards the upkeep of the institute. Since 1930 this sum has been £3,200 a year. Australians, like people of many other countries perhaps, are apt to take for granted established bodies and institutions, particularly if they are efficiently run and are doing good work. A great deal of the original research which is making Australian workers known overseas has come from the Walter and Eliza Hall Institute, and, lest the medical profession be unappreciative, the appearance of the Director's twentieth annual report for the year 1938-1939 is an event to be noted. The first Director, Dr. S. W. Patterson, was appointed in 1919, and Dr. C. H. Kellaway, so well known to readers of this journal by his writings, succeeded him in 1923. The work of the institute is undertaken under conditions which are at once an advantage and a disadvantage. The routine services of the Royal Melbourne Hospital in bacteriology and clinical pathology are carried out at the institute—during the last year 18,479 diagnostic investigations for 11,378 patients were carried out; more than 500 basal metabolic rate estimations were made; 913 electrocardiograms were taken; and 467 autopsies were performed, biopsy sections numbering 1,510 from 1,194 specimens, and 703 sections being taken from autopsy material. Work of this kind must be a distraction to the Director and to certain of his research workers; but it does bring the hospital, as it were, into the institute, and this close association cannot fail to be a stimulus to the members of the honorary staff.

Pride of place is given in his report by the Director to the work of Dr. F. M. Burnet and other members of the virus department. The present is the last of a period of five years during which the work of this department has been financed by annual grants of £1,000 from the Rockefeller Foundation and of the same amount from the Commonwealth Department of Health. For the next five years the institute has received a generous donation of £1,000 a year from Mr. A. E. Cato. With the support of the National Health and Medical Research Council this virus investigation will be continued for a further term. Among the conditions investigated in the virus department during the past twelve months have been poliomyelitis, herpes, influenza, "Q" fever, psittacosis and myxomatosis. Sixteen important papers have been published—six in this journal and six in *The Australian Journal of Experimental Biology and Medical Science*. In other words, twelve of the sixteen papers have been published in the Commonwealth. In the department of physiology work has been done by Dr. W. Feldberg, Dr. E. R. Trethewie and the Director on tissue injury by snake venom, staphylococcal toxin, mercuric chloride, radiant energy, anæsthetics, anaphylaxis, and so on. Ten papers have been published or are in the press, seven in Australia. From the biochemical department three papers have been sent to *The Australian Journal of Experimental Biology and Medical Science*. In the field of clinical research work has been done by Dr. Ian J. Wood, who has sent one paper to this journal and one to *The Australian and New Zealand Journal of Surgery*. It need hardly be stated that the published papers do not represent all the work that has been going on at the institute. Much work is in progress, and the results will doubtless be published in due course. The financial statement shows that this work has all been carried out for the modest sum of £5,399. The Director, Dr. C. H. Kellaway, is to be congratulated on his report, and he and his co-workers will continue to enjoy the confidence and goodwill of the medical profession in Australia.

OBESITY AND HYPERTENSION.

THE relationship between obesity and arterial hypertension has always excited the interest of clinicians. Many extensive surveys have shown that in health blood pressure tends to rise with an increase in body weight. J. E. Wood and J. R. Cash state that no experiments have been undertaken to show the effect of a rapid gain in weight on the blood pressure, and they have made observations on the blood pressure of normal dogs and of dogs with experimentally produced hypertension.[1] They have found that systolic blood pressure in both types of animal rises with a gain in weight and falls when weight is lost; the diastolic pressure varies but little. Their observations, they state, do not indicate that obesity is a cause of essential hypertension, but support the idea that overweight may be a factor of importance in the elevation of systolic blood pressure. Their paper is worthy of careful study.

[1] *Annals of Internal Medicine*, July, 1939.

Abstracts from Current Medical Literature.

BACTERIOLOGY AND IMMUNOLOGY.

Antibodies in Swine Influenza.

CARLOS T. ROSENBUSCH AND RICHARD E. SHOPE (*The Journal of Experimental Medicine*, April, 1939) devised experiments to determine the time following infection when antibodies first appear in the serum and when they reach their highest titre. Six swine were bled and then inoculated with a mixture of influenza virus and *Hæmophilus influenzæ suis*. In four animals typical attacks of swine influenza developed, and in two there occurred modified forms, which the authors called "filtrate disease", resembling that which develops when the animals are inoculated with virus only. They were bled daily during illness and after recovery. The specimens of serum thus obtained were titrated for the presence of antibody content by neutralization tests on white mice, infected mouse lung emulsion being used as virus, mixed in equal parts with the dilution of swine serum to be tested. It was found in the serum of the four swine which developed the typical disease lasting seven days, that the antibodies appeared on the seventh day and reached their maximum titre on the fifteenth to twentieth day after infection, when the animals had recovered. In the two swine with inapparent infections the antibodies were slower to appear. There was no correlation with the height of antibody titre and the severity of the disease; but there was a correlation between the appearance of antibody in the four typical cases and the cessation of clinical symptoms. In the two mildly ill animals, in which signs of infection were present for two days only, antibodies did not appear for many days after improvement in their condition; this suggested that antibodies were not the only cause of clinical recovery.

Immunity in Tuberculosis.

MAX B. LURIE (*The Journal of Experimental Medicine*, April, 1939) has studied the mobilization of mononuclear phagocytes in normal and immunized animals and their relative capacity for division and phagocytosis. He continued to work with the subcutaneous agar focus containing tubercle bacilli, and examined the cellular content of exudates from non-specific irritants produced by injection of 3% starch and aleuronat into the pleural cavities of normal and immunized animals. He found that in tuberculous and vaccinated guinea-pigs and rabbits mononuclear phagocytes were mobilized at the site of inoculation of a non-specific irritant much more rapidly than in the normal animal. The pH at the site of inoculation was slightly lower than in the normal animal, but bore no constant relationship to the mobilization of phagocytes. The rate of division of these mononuclear cells was greater than in the normal control. Experiments were devised to test this phagocytic power *in vitro*, carbon particles, staphylococci, and tubercle bacilli being used as the test material for phagocytosis; and it was always found that the cells from the actively tuberculous animals were more rapid in phagocytosis than those from the normal animal. The enhancement of phagocytic capacity found in animals vaccinated with an organism of low virulence was also of a low order. When the tests *in vitro* were set up in serum from either normal or tuberculous individuals, the increased activity of the cells from the tuberculous animal still persisted. The method of action of these cells seemed to be due to some increase in physiological activity rather than to an extracellular factor, for the discovery of which the experiments had been carried out.

MAX B. LURIE (*The Journal of Experimental Medicine*, April, 1939) has continued his studies in immunity in tuberculosis. He studied the role of extracellular factors and local immunity in the fixation and inhibition of the growth of tubercle bacilli in guinea-pigs and rabbits. He used both normal and sensitized animals, and again used the method of inoculation by an agar mass impregnated with tubercle bacilli. He found that in guinea-pigs the bacilli were more effectively fixed at the portal of entry than in rabbits, and he correlated this with the greater sensitivity of the guinea-pig to tuberculosis and the larger amount of tissue damage at the site of injection. He has adduced histological evidence of the fineness of the fibrinogen network, thrombosis of the adjacent lymphatic vessels, and also of a greater concentration of antibody. He believes that non-specific factors were chiefly involved, as in the guinea-pig trypan blue or India ink particles concentrated at the site of injection better than in the rabbit. The author prepared a series of collodion sacs, which were proved impermeable to cells, inoculated them with the agar mass containing tubercle bacilli, and placed them in the peritoneal cavity of the test animals; he removed them after fourteen days and investigated the effect of the extracellular factors on the growth of the bacilli. He found that cell-free fluids would support the growth of the bacilli, and the normal animal produced a thick cellular tissue membrane about the sac, within which the growth of bacilli went on apace in the outer layers. In the tuberculous animal, on the other hand, there was only a small coating of cells about the sac, while the fluid which permeated to the interior had had a bacteriostatic effect, there being only small increase in numbers of bacilli. He also examined the appearance of the colonies of bacilli grown under these conditions, and found that there was frequently a non-acid-fast centre, from which the mass sprouted acid-fast rods.

Tubercle Bacilli in the Gastric Juice.

R. H. STIEHM (*The American Journal of the Medical Sciences*, April, 1939), examining fifty students suffering from pulmonary tuberculosis of minimal extent discovered radiologically, could not find tubercle bacilli in the sputum (which was usually just saliva) of any; but tubercle bacilli were found in stained films made from the centrifuged gastric juice of nine of the students, while the gastric juice of 36 of the students produced tuberculosis in guinea-pigs. Three students with pleural effusion all had tubercle bacilli in their gastric juice, as proven by the inoculation of guinea-pigs.

Droplet Infection.

W. H. BRADLEY (*Journal of the Royal Sanitary Institute*, March, 1939) discusses the prevention of droplet infection, with special reference to streptococcal disease. He suggests that most of the notifiable fevers are spread by droplets, and that consequently most of the disabling chronic diseases of later life are initiated in this way. He discusses the greater knowledge of streptococci consequent on the Lancefield grouping and the Griffiths typing, and observes that if another disease was passing through a community and an invading streptococcus was found as a secondary infection, clinical study would show the progress of the invader as a distinct episode with an infecting course of its own. Detailed descriptions of the appearance of the tonsils in epidemics of sore throat due to different types, followed by the development of rheumatism, were taken, and it was noted that in the presence of a rash the appearances of the throat might be considerably modified from that usually described as a follicular tonsillitis. The author notes the points in evidence of the importance of droplet infection, namely, seasonal incidence, bed-to-bed infection in wards and dormitories, cross-infection in scarlet fever wards between different types, the spread of puerperal fever in maternity hospitals, the increased attack rate associated with catarrhal infections, and the control of spread by spacing and the use of masks. He also discusses the proof afforded by the use of the Wells air centrifuge in a room where a boy with a Type VI streptococcus infection was being nursed. Many colonies of organisms were grown from air obtained from within a yard of the boy's head, few from air five feet away, and none from ten feet distant. The author holds that the education of the public in the importance of the isolation of early sufferers from sore throat would be helpful, and that early

detection of a new type of infection in a community should be attempted. The identification of representative organisms from each wave of infection in a district would make possible a distinct advance in prevention. The author urges that fresh air is the best insulator, with freedom from dust and ample sunlight in addition. In conclusion he suggests that the possibility of a general catalogue of the coccal flora of communities, exemplified in Gordon's early work on meningococci, might be considered in times of threatened mobilization or war, where herd assembly, the sudden establishment of new communities or mass assemblies would find droplet infection their greatest enemy.

HYGIENE.

Specific and Non-Specific Serum Treatment of Scarlet Fever.

F. H. TOP AND D. C. YOUNG (*The American Journal of Public Health*, May, 1939) have studied the therapeutic effect of scarlet fever convalescent serum, scarlet fever antitoxin and a non-specific serum in 995 moderately severe cases in hospital. A single dose of one of the types of serum was given to patients in rotation. The need for augmented treatment, the temperature pattern and the occurrence of complications were used as tests of efficiency in action. Non-specific serum proved valuable, but specific serum was more useful.

Neonatal Mortality by Order of Birth and Age of Parents.

J. YERUSHALMY (*American Journal of Hygiene*, September, 1938) has analysed more than 82,000 births in the State of New York in 1936 in regard to the order of birth and age of parents in relation to neonatal mortality. Only resident mothers and infants whose birth and first month belong to 1936 were included. Of 82,140 born, 2,563 died under one month of age, or 31‰. Second births show the minimum neonatal mortality figures. First births are 24% in excess. From the fifth birth a sharp rise occurs, and births ten and over have over twice the rate. Boys give a considerably higher rate than girls, with a minimum at the third birth. Illegitimate births (2·3%) showed double the ordinary rate, but first births seemed the safest. Plural births (2·14%) represent four times the neonatal rate. Premature births (4%) form the major problem, their neonatal mortality being more than 25 times as high as that of full-term children; 39% did not live to be one month old, the rate being lowest for first births. The physician's finding of prematurity appears to correspond to the weight criterion. Full-term babies have a neonatal mortality of just about half the average, 15‰, but the figure for the first born was 45% in excess of the second born. Prematurity was more frequent in urban groups and for boys (44‰), more frequent still in illegitimates (87‰), and most of all in plural births (259‰). Stillbirths are about as frequent as neonatal deaths and showed similar frequency in birth order. Premature infants gave the high rate for stillbirths of 249‰, as against 15‰ for full-term babies. In the whole group examined, prematurity supplied 51% of the first month deaths. The maximum of survivors belong to the second births, the first born equalled births numbers 5 to 7, and fewest survived in the births of highest order. Neonatal mortality is lowest among the children of mothers of average age twenty-seven to twenty-eight years, and for those of both youngest and oldest mothers is double. Prematurity tends to follow the same pattern. Stillbirths, however, do not change till they rise after thirty years, where the age of the mother here dominates. The combined losses at birth in the first month are least in the offspring of mothers of twenty to twenty-three years. An optimum interval exists between successive births; the neonatal mortality is higher both in young mothers with frequent children and in old mothers bearing late and with births spaced far apart. Independent of the mother's age, an effect of the father's age can be demonstrated in neonatal mortality. The optimum figures are for fathers twenty-five to twenty-nine years, and high for young fathers. Parents of equal ages show up best, fathers younger than the mothers worst.

Methæmoglobinæmia and its Measurement.

D. O. HAMBLIN AND A. F. MANGELSDORFF (*Journal of Industrial Hygiene and Toxicology*, October, 1938) introduce a description of their work with a brief general discussion of the occurrence of methæmoglobinæmia in acute poisoning by various substances, such as acetanilid, sulphanilamide, drugs of the nitrite group, the nitro and amino derivatives of the aromatic series, such as aniline oil, nitrobenzene, para- and meta-nitroaniline. There is a lack of uniformity of opinion as to what compounds may give rise to identifiable methæmoglobin upon absorption into the human system and as to what concentration methæmoglobin must reach in the blood before it may be identified spectroscopically. Also, as late as 1935, there was still considerable confusion regarding the spectral appearance of methæmoglobin. The present workers in their method used a General Electric automatic recording spectrophotometer and 0·1 cubic centimetre of blood diluted and laked immediately with distilled water up to 2·5 cubic centimetres. This solution is transferred to the glass cell of the instrument and a curve is recorded. Standard curves of oxyhæmoglobin were made and a composite of 25 such curves was adopted as the standard 100% oxyhæmoglobin curve. The 100% methæmoglobin standard solution was prepared according to a method based on that described by other workers. Standard methæmoglobin curves were prepared and a composite of these was adopted as the standard 100% methæmoglobin curve. The authors state that with the method described it is possible to obtain in ten minutes an accurate report on the percentage of methæmoglobin present in any blood sample. In every case of poisoning investigated the severity of the clinical picture has paralleled the concentration of methæmoglobin present.

A Ten Years' Trial of Vaccination with the "B.C.G."

C. NAESLUND (*Revue de la Tuberculose*, June, 1939) reports that in the province of Norrbotten, in Sweden, during the period of ten years ending in 1937, 13,103 new-born children were orally vaccinated with the "B.C.G." (bacillus of Calmette and Guérin); amongst these the number of deaths from all causes was 754, or 6·10%, and from tuberculosis 18, or 0·15%. The number of children born but not vaccinated was 34,845; amongst these the number of deaths from all causes was 3,501, or 11·17%, and from tuberculosis 199, or 0·63%.

The Teeth as "Indicators" in Tuberculosis.

G. E. KING-TURNER (*Tubercle*, April, 1939) suggests that the occurrence or recurrence of dental decalcification or caries in persons suffering from tuberculosis or in "contacts" is a danger signal, indicating a need for specially close and frequent observation. When pyorrhœa and tartar occur, on the other hand, or when dental caries becomes spontaneously arrested, the conditions appear to be favourable for the calcification of tuberculous lesions.

The Treatment of Scabies.

D. V. CURREY (*Canadian Public Health Journal*, June, 1939) states that in Ontario various methods have been in use for the treatment of scabies in school children. When the old sulphur treatment was used the average absence from school was three weeks, with the Danish ointment the period was ten days; both methods are messy and dermatitis may occur. The use of benzyl benzoate lotion (from Copenhagen) has reduced absence from school to three days. The author recommends an anhydrous 20% soft soap with isopropyl alcohol and benzyl benzoate, equal parts of each. After a thorough hot bath and soaping, and while the patient is still wet, the solution is painted all over; the child is allowed to dry and is repainted. The same clothing and bed linen are used till twenty-four hours later, when after a cleansing bath he puts on a complete change. All other children and even the parents receive the same treatment at the same time. Of 400 patients, only 12 required a second treatment, and the cost is low.

British Medical Association News.

MEETING OF THE FEDERAL COUNCIL.

A MEETING of the Federal Council of the British Medical Association in Australia was held at British Medical Association House, 135, Macquarie Street, Sydney, on September 6 and 7, 1939, SIR HENRY NEWLAND, the President, in the chair.

Representatives.

The following representatives of the Branches were present:

New South Wales: Dr. George Bell, O.B.E., Dr. W. F. Simmonds.

Queensland: Dr. D. Gifford Croll, C.B.E., Dr. T. A. Price.

South Australia: Sir Henry Newland, C.B.E., D.S.O., Dr. A. F. Stokes.

Victoria: Dr. F. L. Davies, Dr. H. C. Colville.

Western Australia: Dr. F. W. Carter, Dr. Noel Cuthbert (as substitute for Dr. L. E. Le Souef).

Dr. George Bell acted as proxy for Dr. S. Gibson and Dr. W. E. L. H. Crowther.

Minutes.

The minutes of the previous meeting of the Federal Council of March 14, 1939, which had been circulated among members, were taken as read and signed as correct.

Appointment of General Secretary.

The General Secretary, Dr. J. G. Hunter, whose appointment was due to terminate at the end of September, 1939, was reappointed for a further period of two years, on the motion of Dr. George Bell, seconded by Dr. T. A. Price.

Annual Report of the Federal Council.

The annual report of the Federal Council for the year ended June 30, 1939, was received and adopted.

Finance.

The financial statement as at June 30, 1939, was presented. The statement included the Federal Council account and the Australasian Medical Congress (British Medical Association) fund account. The statement was received.

The General Secretary, in discussing the Federal national health insurance emergency account, pointed out that a considerable sum of money was still owing to the legal advisers of the Federal Council in connexion with the Royal Commission on National Health Insurance, and that the amount in hand was not sufficient to meet the liability. He gave figures setting out the amounts which had been paid into the central fund by each of the Branches, and said that if the Branches were to pay to the central fund the moneys that they had in hand, the liability would be met. He explained that in some States it had been necessary to spend more money in collecting evidence for the royal commission than in others, and for this reason some Branches had only small sums available. It was resolved, on the motion of Dr. A. F. Stokes, that each Branch should be asked to pay its surplus to the central fund.

In discussing the financial position of the Federal Council, the Secretary pointed out that in accordance with instructions received at the previous meeting, he had written to the Council of the Parent Body, explaining the present financial state of the Federal Council. He had received two replies from the London office. In his latest communication Dr. C. Hill, Deputy Secretary, had forwarded for the consideration of the Federal Council a proposal which had been adopted by the council at its meeting in Aberdeen to meet the needs of the South African Branch of the Association. According to this proposal, the receipt of *The British Medical Journal* by members of the Association in Australia would not be obligatory. Instead of sending to England the sum of 25s. 6d. in respect of each member, the Branches would then send a *per capita* payment of 5s. If this scheme were adopted, THE MEDICAL JOURNAL OF AUSTRALIA would be regarded as the official organ of the British Medical Association in Australia, and it would be asked to undertake the publication of such notices and communications as might be sent to it from the Parent Council. The Parent Body would undertake to send to each member a copy of the annual report of council and of the financial statements. Any member in Australia who wished to receive *The British Medical Journal* would be able to do so on the payment to the Home Association of £1 11s. 6d., English currency. In discussing this proposal, the General Secretary said that it was his impression that this proposal would not be generally acceptable to Australian members. He thought that members would expect their subscriptions to the Association to be reduced by the amount of £1, that is, the difference between the present *per capita* payment of £1 5s. 6d. and the proposed payment of 5s. It would, of course, in these circumstances, be impossible for the Federal Council to continue its activities. Moreover, a member who wished to receive *The British Medical Journal*—and these, in his opinion, were many—would have to pay practically £2, Australian currency, each year. He did not think that this would be acceptable. Members of the Federal Council had before them the financial statement of the Parent Body, as published in *The British Medical Journal*, and the Secretary pointed out that the amount expended on its postage was almost equal to the sum which had to be paid from the general funds of the Association into the journal account. In other words, *The British Medical Journal*, with its circulation amongst 38,030 members, was practically self-supporting if the sum expended on postage was taken into consideration.

The view was expressed that the question was not one which could be dealt with immediately, either by the Federal Council or by the Branch councils, but that it would have to be referred to the general body of members.

The General Secretary said that the New South Wales Branch would discuss an increase in the annual subscription. The South Australian Branch had increased its subscription by ten shillings, and both the Western Australian and Queensland Branches had already increased their subscription.

Dr. T. A. Price said that the Queensland Branch had recently found it necessary to increase its subscription by two guineas a year. This had been necessary on account of certain problems affecting the Queensland Branch. The council had expected a certain amount of opposition to the proposal, but none had arisen. Dr. Price expressed the opinion that the work of the Federal Council was worth while and that its activities should not be interrupted through lack of funds. He wondered whether members of the Australian Branches realized that they received all the advantages of membership for a sum which was less than that paid by a good trades unionist to his union.

Dr. George Bell said that he regarded *The British Medical Journal* as an important imperial tie, and he held that nothing should be done which might even suggest a weakening of the bonds between Australia and the Home Country.

Dr. H. C. Colville asked whether the proposal would be a financial advantage to Australian members. Dr. George Bell, in reply, said that he did not think it would, and he thought that the matter should be deferred until the Branches had time to consider it. Both Dr. Price and Dr. Noel Cuthbert thought that no move should be made in the matter until after the end of the war.

Sir Henry Newland said that the position had altered since 1932, when he and Dr. J. Newman Morris had discussed the question of Federal Council finance with the Council of the Parent Body in England. The proposal had at that time been made that the taking of *The British*

Medical Journal should be made optional to Australian members. The Council of the Parent Body had replied that it would be impossible for them to accede to this request, since they could not differentiate between members of the Branches in different parts of the world. He thought that the arrangement suggested for the South African Branch showed that the Parent Council had changed its mind and was now prepared to differentiate. He wondered whether it would not be possible for the Parent Association to reduce the *per capita* payment for overseas members from 25s. 6d. to 21s. *per annum*. If this reduction were made there would be no need for the £1,000 which had been granted by the Parent Body to the Federal Council for three years.

On the motion of Dr. Noel Cuthbert, seconded by Dr. F. W. Carter, it was decided that consideration should be deferred until the Branches had had an opportunity of considering the proposals, and that the suggestion should be made to the Parent Body that the *per capita* payment might be reduced from 25s. 6d. to 21s.

The immediate requirements of the Federal Council were then considered. In reply to a member the General Secretary said that immediate needs would be met if the Federal Council received the sum of 6s. per member from the Branches for the current year. In other words, the Branches would require to add another 4s. It was resolved that the Branches should be asked for this amount.

Medical Officers' Relief Fund (Federal).

The report of the trustees of the Medical Officers' Relief Fund (Federal) for the year ended June 30, 1939, was presented by Dr. George Bell. Dr. Bell stated that twelve loans were in existence on June 30, 1939, as against thirteen at the end of the previous year. The sum of money on loan had been reduced by £322, and the assets had been a little more than correspondingly increased. In reply to a question by Dr. F. L. Davies, Dr. Bell stated that the fund could be used only for the relief of medical officers disabled during the last war. The fund had been subscribed by members of the medical profession. If anything of the sort was needed in the present war another fund would have to be created. The report was adopted.

The Reporting of Medical Conferences.

A letter was read from the Victorian Branch, in which a proposal was made that the Federal Council should have a shorthand writer to report the proceedings of any conference in which the medical profession was represented. Dr. F. L. Davies, in discussing this letter, expressed the opinion that the medical profession should have had a reporter at the recent conference with Sir Frederick Stewart. Sir Henry Newland explained that when national health insurance was discussed with Mr. Casey, who was then Commonwealth Treasurer, Mr. Casey would not allow a reporter for the medical profession to be present. Dr. Davies commented on the obvious unfairness of this attitude, and Dr. Noel Cuthbert suggested that the words "whenever possible" should be included in a motion based on the suggestion of the Victorian Branch. This was adopted.

New Zealand Ophthalmologists.

A letter was read from the Ophthalmological Society of Australia (British Medical Association) in reference to a proposal that ophthalmologists in New Zealand should be allowed to join the society. It was realized that as at present constituted the Ophthalmological Society could not admit members from New Zealand. The New Zealand ophthalmologists wished to join and thought that the name of the society might be altered to allow of their membership. Sir Henry Newland pointed out that this could not be done, and during discussion it became clear that the members of the Federal Council wished to make some arrangement which would be acceptable to New Zealand ophthalmologists. It was thought they might be elected as honorary members of the Ophthalmological Society of Australia, or else that they might form a small group of their own and apply for affiliation with the Australian body. The Federal Council was quite agreeable to such affiliation if the ophthalmologists thought it desirable. It was resolved that the views of the Federal Council should be communicated to the Ophthalmological Society of Australia.

Catgut.

A letter was received from the Director-General, Commonwealth Department of Health, inquiring whether the Association had advised its members to make certain that any sutures they used carried a certificate of sterility from some properly authorized body. The Director-General was informed that the New South Wales Branch had published an extract from a report by the Section of Surgery of that Branch in regard to the preparation of catgut for surgical purposes. In this report the section expressed the opinion that it was essential that an adequate sample of each batch of prepared gut should be submitted to the bacteriological test prescribed under the regulations of the *Therapeutic Substances Act* of Great Britain at the hands of a competent bacteriologist and that in the absence of such a test surgeons should buy sterilized catgut certified under the English *Therapeutic Substances Act*.

The Prevention of Deafness.

A letter was read from the Director-General, Commonwealth Department of Health, in which he stated that the National Health and Medical Research Council proposed to undertake a general investigation in regard to the prevention of deafness. The Director-General invited the Federal Council to nominate a subcommittee of three, who would act with the National Health and Medical Research Council. The Secretary reported that he had communicated with the President, and that as the National Health and Medical Research Council was to hold its meeting in Sydney, Dr. D. G. Carruthers, Dr. E. P. Blashki and Dr. E. McA. Steel were appointed.

Members of the Association at Darwin.

A letter was received from the Western Australian Branch, stating that a member of the Branch had been transferred to Darwin. As there was no Branch of the Association at Darwin, the Western Australian Branch wished to know to which Branch the transferred member would belong. The Western Australian Branch was informed that members of the Association at Darwin retained their membership of the Branch in the area from which they had moved.

Hospital Policy.

A letter was received from the Queensland Branch with regard to a hospital policy for Australia. It was decided that in view of the international situation the time was inopportune for a discussion of this subject.

National Health Insurance in Canada.

A letter from the Canadian Medical Association, setting out in detail the progress of events in British Columbia relating to national health insurance, was received.

Wiluna Medical Fund (Western Australia).

A letter from the Western Australian Branch, dealing with medical practice at Wiluna, was received. A communication from the Western Australian Branch on this matter was published in THE MEDICAL JOURNAL OF AUSTRALIA on September 16, 1939.

Congress of the International Hospital Association.

A letter was received from the Department of Commerce of the Commonwealth announcing that the sixth biennial congress of the International Hospital Association would be held at Toronto, Canada, on September 19 to 23, 1939, and suggesting that representatives from Australia should be appointed. As the communication had been received too late no action was taken.

Australasian Medical Congress (British Medical Association).

Fifth Session.

Further reference was made to a letter received at the last meeting of the Federal Council from the Australasian Medical Publishing Company, Limited, suggesting that it might be reimbursed for its loss in connexion with the publication of papers read at the fifth session at Adelaide. At the last meeting it had been decided that consideration should be deferred until the financial arrangements in connexion with the fifth session had been discussed with the executive committee of that session. The General Secretary reported that he had communicated with the executive committee, and he read a reply in which it was explained that the arrangements for publication had been made with the Editor of THE MEDICAL JOURNAL OF AUSTRALIA when he had visited the Branch before congress was held. Three possible methods of publication had been discussed with the Editor, and the method finally adopted had been chosen as that which would involve the least expenditure. After discussion it was resolved that the Federal Council should accede to the request of the Australasian Medical Publishing Company, Limited.

Sixth Session.

After the General Secretary had reported the arrangements so far completed for the holding of the sixth session of congress at Perth in September, 1940, it was resolved that in view of the outbreak of war the date of congress should be cancelled and that the executive committee should be asked to suspend its activities.

Organization of the British Medical Association in Australia.

Further reference was made to the organization of the British Medical Association in Australia, which had been discussed at the last meeting of the Federal Council. At that meeting consideration had been deferred in view of the fact that complete autonomy could not be granted to the Australian Branches by the Parent Body until each Branch had become a corporate body. The Victorian Branch explained to the Federal Council the difficulties associated with incorporation of the Victorian Branch. In view of the declaration of war further activities in this direction were suspended.

The Voting Power of Branch Representatives on the Federal Council.

A letter was received from the Victorian Branch suggesting that the voting power of representatives of the Branches at the Federal Council should be altered so that Branches with a large membership might have a voting power more in accordance with their membership than was at present the case. After discussion the letter was received.

Election of Branch Representatives on the Federal Council.

The Secretary stated that a notice of motion had been received from the South Australian Branch to the effect that Branch representatives on the Federal Council should be elected by members of the Branch and not by the council. This motion had been sent to the Branches. The New South Wales, the Queensland, the Victorian, the Tasmanian and the Western Australian Branches had all replied, and had stated in effect that they were satisfied with the present method of election by Branch councils. Several members of the Federal Council pointed out that if members of the Federal Council were appointed by Branch members it was possible that persons with no experience of Branch council matters might be elected. Without such experience no Branch member was likely to be of much use in the deliberations of the Federal Council. Sir Henry Newland, on behalf of the South Australian Branch, proposed the motion and Dr. A. F. Stokes seconded it. The motion was lost.

Medical Education.

A letter was received from the New South Wales Branch suggesting that a committee of members of the Association from the Branches in States having medical schools should be formed to consider the question of medical education in Australia. It was pointed out that the British Medical Association in England had issued a report on medical education, and it was thought that the Branches in Australia should collectively make some pronouncement on the subject. The General Secretary said that the New South Wales Branch had considered the matter and had laid down certain general principles for his guidance in regard to certain inquiries that were being made. After discussion, in which several members expressed the view that the suggestion of the New South Wales Branch was valuable, it was decided to take no action on account of the declaration of war.

Doctors' Signs and Nameplates.

A letter was received from the Queensland Branch about the standardization of regulations in regard to doctors' signs and nameplates. It was explained by the Queensland representatives that the Queensland Branch desired to have some uniformity in the rules. The rules of the New South Wales Branch were produced and the sections relating to signs and nameplates were read. It was recommended that these should be adopted so that uniformity might be obtained.

Pensions for Sufferers from Early Tuberculosis.

At its previous meeting the Federal Council received a letter from the South Australian Branch asking it to approach the Federal Government in order to seek adequate pensions for early sufferers from tuberculosis in the several States. The opinion was expressed that the pension was not given to a sufferer from tuberculosis until it was too late, and that even when all possible help was given the amount was insufficient.

Dr. D. G. Croll said that a great deal of the money which was paid to returned soldiers suffering from tuberculosis was wasted, because there was no adequate supervision of the way in which the money was used. He then referred to the wisdom and economy of a patient of his acquaintance, who was in receipt of 17s. 6d. a week, and who lived in a little hut for which he paid 1s. a week rent. He owned a goat, which supplied him with milk, and he was doing very well on his pension. Dr. Croll expressed the view that it was useless to increase pensions unless there was some means of policing them.

Dr. F. W. Carter pointed out that it was necessary that some provision should be made for the family of a tuberculous patient. The patient knew that if his condition was declared to be tuberculosis he would have to leave his work. He therefore kept on working much longer than he should, and his disease inevitably advanced. If the patient knew that his family was provided for, his mind would be relieved of an intolerable anxiety.

Dr. W. F. Simmonds thought that the matter was of Federal importance and should be brought to the notice of the Federal Minister for Health.

In the general discussion which followed, more than one member expressed the opinion that some form of family endowment should be instituted while the patient was undergoing treatment, and that it was inadvisable to give money. After reference to resolutions carried at the plenary session of the Adelaide congress in 1937 it was resolved, on the motion of Dr. F. W. Carter, seconded by Dr. D. G. Croll:

> That this council is of the opinion that the economic factor is of the utmost importance in the control of tuberculosis, and in order (*a*) that the sufferer be induced to submit himself for examination and treatment at the earliest possible moment and (*b*) that the resistance of contacts may be sustained and (*c*) that the anxiety of the breadwinner regarding the economic welfare of his dependants be lessened, a system of family

endowment be introduced by the Government which will enable early curative treatment to be instituted and the families of tuberculous patients to be adequately provided for.

The Koch Method of Cancer Treatment.

At its previous meeting the Federal Council considered a letter from the Western Australian Branch regarding the Koch method of cancer treatment. At that meeting it was decided that the Commonwealth Department of Health should be approached with the suggestion that it might possibly undertake an investigation of the method. The General Secretary reported that he had had from the Director-General of Health a reply in which reference was made to the discussion of the Koch method of treatment at the Cancer Conference, to the investigation of the cure undertaken in New Zealand and to work overseas. The conclusion was that the cure was of no value and that much useful time might be lost by the submission of patients to it. The Director-General of Health did not propose to take any action.

Alien Medical Practitioners.

A letter was received from the Western Australian Branch in regard to the influx of alien practitioners into Australia. It was stated that probably during the next three years 15,000 refugees would come to the Commonwealth, and that a proportion of these would be medical practitioners. Quite a number were already trying to secure registration in several States, and some had succeeded in doing so. The Western Australian Branch was not in favour of the granting of registration for practice in a zone. The failure of the authorities to secure practitioners for these areas was a matter of general living conditions and salary. In the discussion which followed reference was made to the calling up of many medical practitioners for active service in connexion with the war, and it was resolved:

> That in view of the international situation the Federal Council requests the Commonwealth Government to take steps to protect the practices of men called up for service in the armed forces and for attendance on the civil population by stopping the influx of alien practitioners.

Coordination of the Medical Services of the Commonwealth.

Sir Henry Newland was elected as the representative of the council on the Central Executive Committee for Coordination of Medical Services.

War Emergency Organization of the Medical Profession.

The Federal Council discussed at length the war organization of the medical profession in Australia. It also discussed the protection of the practices of men who volunteered or were called up for active service. The following motion was carried:

> The British Medical Association in Australia will do all in its power to cooperate in the defence measures adopted by the Federal Government.

The Formation of Sub-Groups.

The Ophthalmological Society of Australia (British Medical Association).

The Secretary stated that he had received the first annual report of the Ophthalmological Society of Australia (British Medical Association). This was formally received.

Special Group for the Study of Rheumatism.

Further reference was made to the formation of a special group for the study of rheumatism. The President stated that he had received a letter signed by Dr. E. Britten Jones, Dr. A. R. Southwood and Dr. C. B. Sangster, of Adelaide, requesting approval of the formation of a special group, to be known as the Australian Association of Rheumatism and Physical Medicine. He had given formal approval to its formation. The opinion was expressed that in all probability nothing further would be done in the matter at present.

The Public Medical Services.

At previous meetings of the Federal Council consideration had been given to the remuneration of medical officers in the public medical services. A report was presented from the Victorian Branch. This had been drawn up by one of the members of the council, and it was resolved that a copy should be made and forwarded to each member so that it might be considered at the next meeting.

Friendly Society Contract Practice.

The Federal Council discussed the adoption of a model common form of agreement for friendly society contract practice throughout the Commonwealth. It was agreed that it would be advantageous if such an agreement was drawn up and adopted, and it was resolved, on the motion of Dr. George Bell and Dr. A. F. Stokes, that the question should be referred to the Federal Contract Practice Committee, but that all Branches should be asked to take no action at the present juncture.

National Health and Medical Research Council.

It was noted that the appointment of the Federal Council's representative on the National Health and Medical Research Council would lapse before its next meeting. It was resolved that Dr. J. Newman Morris should be reappointed as the Federal Council's representative.

National Health Insurance.

The Secretary read a letter from the Western Australian Branch taking exception to the appointment by the Victorian Branch to the Contract Practice Subcommittee of a member who was not a general practitioner. The Western Australian Branch expressed the view that this subcommittee was intended to be a committee of general practitioners, and that the Victorian Branch appointment did not fulfil the objects for which the committee had been created. Dr. F. L. Davies said that as President of the Victorian Branch he took strong exception to the Western Australian letter. He asked the General Secretary to read the minute dealing with the objects of the subcommittee, and pointed out that the Victorian Branch had been quite in order in making its appointment. It had chosen a man who in its opinion was best calculated to represent the general practitioners in Victoria. After discussion the letter was received.

The period of appointment of the Contract Practice Subcommittee was extended until the next meeting of the Federal Council.

Federal Emergency (Compensation) Fund.

Consideration of the establishment of a Federal emergency (compensation) fund, which had been mentioned at the previous meeting of the Federal Council, was deferred.

Date and Place of Next Meeting.

Determination of the date and place of the next meeting was left in the hands of the President.

Votes of Thanks.

A vote of thanks was accorded to the Council of the New South Wales Branch of the British Medical Association for having provided accommodation for the meeting and for its hospitality.

A vote of thanks was also accorded to Sir Henry Newland for having presided.

SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held in the Medical Society Hall, East Melbourne, on July 5, 1939, DR. F. L. DAVIES, the President, in the chair.

Coronary Occlusion.

DR. T. E. LOWE read a paper entitled "The Pathology of Coronary Ischæmia" (see page 491).

DR. G. A. PENINGTON read a paper entitled "Some Practical Considerations in Coronary Occlusion" (see page 501).

DR. E. S. J. KING congratulated Dr. Lowe and Dr. Penington on their masterly presentation of the subject. He said that he did not think it was generally recognized how much was original in Dr. Lowe's work. If any of the members cared to consult the *Quarterly Cumulative Index Medicus* they would see what a tremendous literature had developed about coronary occlusion. It was a matter for pride that one of the most important advances had been provided by a Melbourne physician. The surgery depended largely on the work of Dr. Lowe; he had demonstrated that the lesion in the heart muscle following a recent coronary occlusion was laminated in character and that an older one became scarred. Dr. Lowe himself had mentioned the work of the Robbs in America, and, though they had investigated the matter, the observations concerning the laminations were the original work of Dr. Lowe. Those observations had a very important bearing on the question of surgery in coronary occlusion. Operation was suggested first by Beck, in Chicago, and not by O'Shaughnessy, as might have been inferred from Dr. Penington's reference to the matter. O'Shaughnessy's contribution had followed, and others had since paid attention to the question. Attempts had been made to improve the circulation by operations on the omentum, lung, pericardium *et cetera* in efforts to establish a new collateral circulation. When the circulation of the heart was normal, the circulatory pressure in the epicardial capillaries was lower than it was in either the omental vessel or the cardiac vessels. The blood passed away from the epicardial capillaries into the veins. Thus, since the passage of blood from one area to another depended on a definite "pressure gradient", a collateral circulation could not develop in that case and the omentum would ultimately lose its attachment to the heart. If the superficial muscles were involved, then a gradient would develop from the omental vessels to the myocardium. It had been shown by Dr. Lowe, however, that in most cases the involved area was deeply situated in the heart wall and the superficial muscle, or a considerable amount of it, was normal; then the gradation of pressure from omentum to the damaged tissue could not occur. It was important that even if a collateral circulation could be established, it was of value only in helping to prevent further damage and could not be of value in affecting muscle already injured. In summing up, Dr. King stated that at present it was not possible to apply the tissue anywhere but at the surface. It was quite obvious that the operation could not in many cases be of any value so far as the supplying of blood to the heart was concerned in coronary occlusion; but he had been astonished at the improvement that had occurred. It could not, however, be due to the establishment of collateral circulation.

DR. C. H. FITTS expressed his appreciation of the papers. He said that a new attitude was being formed towards a common problem. There was no anxiety neurosis about the diagnosis of coronary occlusion. The diagnosis was imperfect, and was usually made only when dramatic pain had supervened. It was like shutting the stable door after the pony had bolted. In the absence of pain diagnosis was difficult. Pericarditis might be a complication of coronary occlusion or it might be isolated. Coronary occlusion could also be present though the electrocardiographic tracings were normal. Dr. Fitts went on to mention the problem of coronary occlusion as a compensable condition in industry; the subject might be in the course of doing his work when pain occurred which proved to be due to coronary occlusion and might prove fatal or cause invalidity. It was of considerable importance to know whether strain or trauma could produce the changes in an apparently healthy individual; the belief that it could brought the subjects before the law courts, and the lack of knowledge added to the difficulty in making satisfactory decisions.

DR. W. W. OSTERMEYER added his congratulations and expressed his conviction that the use of electrocardiography was a definite and dependable method of diagnosis.

DR. F. L. DAVIES, from the chair, thanked those who had contributed to what he regarded as an important discussion of a difficult subject and invited the opening speakers to reply.

Dr. Lowe thanked the members for the reception accorded to his paper. He took the opportunity of acknowledging the encouragement he had received from Professor MacCallum and from Dr. King; they had assisted him to change into a good story what at first had seemed a mere figment of the imagination.

A MEETING of the Section of Neurology and Psychiatry of the New South Wales Branch of the British Medical Association was held in the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, on August 3, 1939, DR. C. G. MCDONALD, the Chairman, in the chair.

The Diencephalon.

DR. A. A. ABBIE read a paper entitled "The Diencephalon: Structural, Functional and Clinical Considerations". This paper was published in the issue of September 16, 1939, at page 421.

The Use of "Cardiazol" in Psychiatry.

DR. BROUGHTON BARRY read a paper entitled "The Use of 'Cardiazol' in Psychiatry". This paper was published in the issue of September 16, 1939, at page 430.[1]

PROFESSOR A. N. BURKITT thanked both speakers for their addresses. In regard to Dr. Abbie's paper, one fact clearly emerged, and that was that the traditional acceptance of the theory associated with the names of Head and Kinnier Wilson, concerning the relationship between the thalamus and the emotions, was now being critically questioned and was in the melting pot.

Recent observations which had some bearing on the problem were: (*a*) that stimulation of the central end of a cut vagus nerve was correlated with increased electrical activity in the orbital region of the frontal lobe of the cerebral hemisphere; and (*b*) the work of Fulton and others upon the premotor and motor cortex had shown that upon excision of various portions of these areas (numbers 4 and 6 of Brodman) ill-defined visceral symptoms, such as constipation *et cetera*, had been found to occur with sufficient regularity to indicate some sort of correlation.

Thus the whole of the frontal lobe, including the vague prefrontal area of various writers, seemed to be definitely related to the viscera and autonomic nervous system, partly by way of the hypothalamus and partly by the dorso-medial nucleus of the thalamus.

Another speculation which Professor Burkitt thought might prove of interest was the fact that in the Australian marsupial mole, *Notoryctes*, the classical bundle of Vicq d'Azyr or mamillo-thalamic tract and the anterior nucleus of the thalamus were extremely diffuse and ill defined. In higher mammals and man this tract was well defined, and the anterior nucleus also. The mamillary body had in recent years been associated with sleep, and it was a curious fact that *Notoryctes* seemed peculiar in its behaviour, in that when in captivity it either exhibited feverish activity or immediately appeared to fall asleep,

[1] Apologies are offered to the Section of Neurology and Psychiatry for the non-publication of the discussion on Dr. Abbie's and Dr. Barry's papers in the issue of September 16.—EDITOR.

with an alteration of its breathing (Wood Jones). Whether there was any correlation between structure and function in the above case would, of course, be only a matter for speculation at present.

In regard to Dr. Barry's interesting address, Professor Burkitt did not feel competent to speak, except to remark that the recent observations upon the frontal lobe which he had mentioned must be taken into account in any treatment, such as "Cardiazol" shock therapy, which aimed at stimulation of the motor area of the frontal lobe.

DR. J. A. L. WALLACE complimented the authors of the two papers read, and thanked them for the very clear exposition of the subjects they had presented.

The two subjects treated of might not at first glance appear directly related, but further research might prove that schizophrenia was related to some disturbance of the thalamus, just as epilepsy seemed to have some association with disturbance of the hypothalamus. With regard to Dr. Barry's idea that refractory wards might not be necessary in the future, owing to the success of "Cardiazol" treatment, he hoped that Dr. Barry was not too optimistic. So far, in the Mental Hospitals Department, under Dr. Wallace's control, four hundred patients suffering from mental disorder had now been treated with "Cardiazol", with excellent results, these results being more promising and more spectacular than even those obtained by the treatment of general paralysis with malaria and inductothermy, which so far had been the greatest advance in the treatment of mental disorders.

DR. OLIVER LATHAM, while expressing his gratitude to Dr. Abbie for his able presentation of new conceptions of the thalamus, also desired to commend the idea of presenting at the same time an anatomical background to a lecture on clinical psychiatry. It was becoming increasingly apparent that the schizophrenic did not regress in a haphazard way. Often the catatonic's strange attitude could be interpreted by the cutting off of influences from some other levels, so that it might be said, "he looked like a thalamic experimental animal", and so on.

Dr. Latham also commended Dr. Barry's strong stand that "Cardiazol" mainly relieved the acute, meaning the insane, manifestations of a patient; it hardly alleviated the weakness in his original mental and/or physical pattern. However, as Reception House records showed that 95% of would-be suicides never made another attempt, so a certain percentage of "Cardiazol"-treated patients would benefit by their contact with new and trained friends and learn better how to apply themselves to life's problems and reverses. To too many men the first encounter created shock. Familiarity bred contempt. Anyhow, the "Cardiazol" treatment broke up continued bad habits of thought: "The silent grief that whispers of the o'erfraught heart, and bids it break."

DR. G. TRAHAIR said that he wished to thank Dr. Abbie for his excellent paper, but regretted that he was not competent to offer any useful comment. In regard to Dr. Barry's paper, however, like Dr. Edwards, he could not share Dr. Barry's optimism about the innocuousness of "Cardiazol" therapy. One danger was that of mental deterioration following convulsive treatment. He had seen one such case, that of a depressed patient who also suffered from arteriosclerosis. Following "Cardiazol" injections the depression had disappeared, but the patient showed some forgetfulness and mild dementia. In this case it seemed probable that the rigors of convulsive therapy in the presence of arteriopathy had caused some actual cortical damage.

DR. A. T. EDWARDS thanked the speakers for their papers and stated that he agreed with Dr. Wallace that probably the condition of the hypothalamus was a factor in the symptomatology of many cases of schizophrenia, and possibly in some forms of epilepsy. He pointed out that encephalograms made of a patient in convulsions induced by "Cardiazol" gave waves in the frontal area, typical of *grand mal*.

He gave the following summary of the results of "Cardiazol" treatment on patients at Callan Park, as set out in the accompanying table.

Group.	Cases.	Remissions.	Discharged Improved.	Improved in Hospital.	Not Improved.
Recent	30	13	8	—	9
Chronic	38	1	5	5	—
Manic-depressive psychosis and confusional insanity ..	20	19	—	1	—

Of 150 consecutive schizophrenic patients admitted to hospital from 1931 onwards and not treated by "Cardiazol", 45% had been discharged "relieved" or "recovered". Of the 30 patients admitted and treated with "Cardiazol", 70% were discharged "relieved" and "recovered".

He agreed with Dr. Barry that sclerosis of the veins was very rare, even though the solution was not buffered.

He disagreed with the statement that it was not necessary to obtain a full *grand mal* type of fit, as experience had shown that "a failed fit" produced agitation for some time and led to increased fear of the injections. He suggested that no effort should be made to shorten the period of apnœa following the fit, as workers in America had suggested that this was an important part of the action of the treatment and had deliberately prolonged this period by holding the nose and mouth.

In regard to dosage, he pointed out that the amount that could be given safely was comparatively immense, 27 cubic centimetres of 10% solution having been given in two minutes without any ill effect. He stressed the possible dangers of fractures during the fit. In a recent issue of *The Journal of the American Medical Association* eleven cases of compression fracture of the vertebræ, three fractures of the pelvis and three dislocated shoulders had been described. Figures obtainable concerning the treatment in Australia suggested that Australian experience had been more fortunate, and at Callan Park only one fracture, that of a humerus, had occurred after some 5,000 injections.

Finally, it was suggested that "Triazol" might be given to those patients to whom it was desired to give more frequent fits than the usual two or three per week.

DR. C. G. MCDONALD, from the chair, said that he had enjoyed a veritable feast of knowledge in the two papers presented, and regretted that the meeting had not been more widely advertised, so that a larger audience would have had the opportunity of hearing them. He congratulated the speakers on their extremely interesting papers, and was fully conscious of the immense amount of work which the preparation of the papers must have entailed.

In reply, Dr. Abbie reiterated his belief that the seat of conscious appreciation of emotion was to be found in the cerebral cortex.

Dr. Barry, in reply to Dr. Trahair, agreed that in the majority of cases it was necessary to produce a major fit; however, undoubtedly there were cases in which a sub-fit dose produced an effect that was not deleterious, as he had quoted previously. He agreed with Dr. Trahair that in the majority of cases it was necessary to follow an unsuccessful injection with another successful one. Dr. Trahair had also quoted a case in which a memory defect occurred after treatment. Dr. Barry replied that he had as yet seen no such cases.

In reply to Dr. Edwards, Dr. Barry said that he had as yet seen no cases of compression fracture of the spine, and indeed had observed no symptoms that would point to such. He agreed that it would be desirable for an electrocardiograph to be taken of each patient before he was subjected to "Cardiazol" therapy, as was the standard practice at some continental hospitals. Unfortunately, however, such desirable refinements as electrocardiographic apparatus were not as yet included in mental hospital equipment in New South Wales.

VICTORIAN BRANCH NEWS.

THE following judgement of the Workers' Compensation Board of Victoria is published at the request of the Council of the Victorian Branch for the information of members, as it is the first decision of the Board on the matter of a claim for compensation for hernia.

Bruce v. the Shire of Hampden, 902/38.

In this matter, the Applicant, Harold Herman Bruce, claims from the Respondent, the President, Councillors and Ratepayers of the Shire of Hampden, compensation on the ground that on the 29th day of September, 1938, he suffered personal injury by accident arising out of and in the course of his employment by the Respondent. The injury alleged was a left inguinal hernia caused, it is said, when the Applicant was at work unloading heavy drums of tar from a railway truck at the Skipton Railway Station. The Respondent's answer to this claim is, in substance, that the hernia of which the Applicant complains was not caused by any accident arising out of and in the course of his employment and that the hernia existed prior to the 29th day of September, 1938.

According to the evidence the association of hernia with trauma is exceptional, and we are satisfied that the hernia of which the Applicant complains is not of the type which has been called true traumatic hernia. The hernia which affected the Applicant at or about the date of the alleged accident was, we conclude, an oblique inguinal hernia.

The latest theory of the origin of such a hernia is discussed in the article by Dr. Wainwright, M.A., M.D., on the Relation between oblique inguinal hernia and workmen's Compensation laws published in the *Archives of Surgery*, Vol. 6, p. 605, and this article was produced in evidence. This theory, known as the saccular theory, in effect amounts to this, that hernia is practically always due first to the presence of a pre-formed sac or open pouch of peritoneum which, in the inguinal variety follows the testis in its descent into the scrotum, which pouch has failed to close in the normal way; and second, to the presence of structural weakness in the neighbourhood of the hernial orifices due to poorly developed muscles or fascia, and it is said that given these all-important anatomical causes which are in themselves sufficient in many cases to constitute a potential hernia, the actual hernia may develop by reason of a great variety of exciting causes: among these may be mentioned the daily increase in intra-abdominal pressure incident to the ordinary routine of life, *e.g.*, straining at stool, coughing, sneezing, lifting, etc.

See Wainwright's article, pp. 623-4.

While it is may be suggested that inguinal hernia has a congenital origin the result of a pre-natal anatomical defect, the question remains whether it can ever be regarded as "an injury arising out of and in the course of a worker's employment".

It may perhaps be true to say that the cause of the formation of a hernia is the existence of a congenital sac which has been gradually enlarging through the strains of everyday life, but this theory does not dispose of the question whether or not it may be an injury for the purposes of the Workers' Compensation Acts.

It is now well settled that an aggravation of a disease may constitute an injury arising by accident out of and in the course of a worker's employment.

In *Oates v. Earl Fitzwilliam's Collieries Co.* 1939 All Eng. Reports, Vol. 2, p. 498 at p. 502, Clauson L.J. said: "In our judgment, a physiological injury or change occurring in the course of a man's employment by reason of the work in which he is engaged at or about that moment is an injury by accident arising out of his employment, and this is so even though the injury or change be occasioned partly, or even mainly by the progress or development of an existing disease if the work he is doing at or about the moment of the occurrence of the physiological injury or change contributes in any material degree to its occurrence. Moreover, this is none the less true though there may be no evidence of any strain or similar cause other than that arising out of the man's ordinary work."

See also *Ward v. Corrimal-Balgownie Collieries Ltd.* 61 C.L.R. 120 at 130.

The development of hernia is substantially due to intra-abdominal pressure, and even though the hernia may be of gradual onset there appears to be no reason why intra-abdominal pressure occasioned by some incident in the course of the work in which a man is engaged should not so affect the development of the hernia as to cause him to be incapacitated. The strain or stress of the work in which a man is engaged may, in our opinion, give rise to such intra-abdominal pressure as to cause the initial descent of the hernia. On the other hand, in the case of a man who runs the constant risk of the descent of a hernia where the hernia has started to descend, the work in which he is engaged may, in our opinion, be the means of causing more bowel or omentum to be forced into the sac so as to bring about an incapacity for work.

We consider, therefore, that a worker is entitled to compensation where he can establish that there is some causal connection between the work which he is doing in the course of his employment and the hernia of which he complains.

That a hernia of which a worker complains may afford him a right to compensation is established by many decisions of the Courts both in England and Australasia.

Claims for compensation for hernial injury must, in each case, be determined by the facts of the particular case, and we consider it undesirable to adopt any of the general criteria which have been suggested as a means of determining whether hernia should or should not be regarded as an injury entitling a worker to compensation.

We now come to the evidence relating to the Applicant's claim. The Applicant, when pulling a drum of tar, slipped, and felt a severe pain in the region of the left groin. He says that he stopped for a few minutes after the pain and felt sick and then better. He continued his work with the Respondent until the 4th day of October when he consulted Dr. Donaldson who discovered that he had a small hernial protrusion at or near the site of the pain. This protrusion appeared when he was standing up and disappeared when he was recumbent. The Applicant says that his pain continued and was such that he could not keep his legs apart. The Applicant said that he never before had any trouble in the groin that he was conscious of, and had never strained himself there before.

We think that at or about the time of the accident the Applicant had a congenital sac, and on this question we accept the evidence of Dr. Upjohn who was called for the Applicant and that of Dr. Balcombe Quick who was called on behalf of the Respondent.

Dr. Upjohn said that nothing that he had heard about the Applicant made him consider that he had anything but an old hernial sac made manifest by a recent strain, while Dr. Quick considered that there was not a narrow entrance to the sac owing to the fact that the hernia was reducible, and he also expressed the opinion that the Applicant had a long existing congenital sac.

The next question is whether the hernial condition of which the Applicant complained was caused or aggravated by the work he did on the afternoon of the 29th day of September, 1938.

Dr. Upjohn said that a hernial swelling produced slowly would be harmless, but if produced suddenly would be incapacitating and according to Dr. Quick if there had been at the time of the alleged strain only a minor entry or addition of abdominal content into the sac, it would not have produced such a condition as would have resulted in any incapacity for work and even if the work had caused an aggravation of a pre-existing hernial condition causing disablement, the Applicant would have been incapacitated for three or four days.

It appears to us on the medical evidence which we consider we should accept, that a hernia produced or aggravated by the work which the Applicant was doing would have not only caused the Applicant intense pain, but would also have prevented the Applicant from doing

the work which he in fact did after he received the alleged strain. Almost immediately after the Applicant received this strain he removed from the truck at which he was working, five drums of tar each weighing about 4 cwt., and he continued his work with the Respondent from the Thursday afternoon when the alleged accident happened until the following Tuesday, doing, as he says, all that he was required to do. If the hernia had been brought about suddenly by the work which the Applicant had been doing on the Thursday afternoon, the Applicant, according to the evidence of Dr. Upjohn, could not have shifted five drums of tar each weighing about 4 cwt. without prostration. As we construe the evidence of Dr. Quick, the incapacity of which the Applicant complains, could only have been caused by an appreciable entry of abdominal content into the sac, or an appreciable addition thereto, and if this in fact occurred, it would not have been feasible for the Applicant to have continued his work.

We, therefore, find upon the evidence that the Applicant has not proved that the incapacity in respect of which he claims compensation was the result of a hernia caused or aggravated by the work he was doing on the afternoon of the 29th day of September, 1938. We therefore make our award for the Respondent. No order as to costs.

(Signed) T. S. CLYNE.

Correspondence.

MEDICINE AND PUBLIC LIFE.

SIR: Your editorial on the subject of the service which medical men can render in Parliament is of much interest. There are numberless social activities which could be materially assisted by the application of trained medical science. But as Lord Horder indicated, there is no place for such men under a rigid party system. Most thoughtful men are not strong party supporters because they see both sides. One respected member of the Victorian legislature informs me that 80% of the bills submitted are not party measures, but become so owing to the party mechanism.

But if the system of proportional representation were utilized, places would be found for them. The system is slowly spreading throughout the democratic world—in fact more rapidly than one would expect, knowing the party obstacles. I have elsewhere set out a list of countries, cities and other organizations which have adopted it. Political outlook is slow to change, but as Galileo remarked on a famous occasion, "*E pur se muove*".

Yours, etc.,

JAMES W. BARRETT.

103-105, Collins Street,
Melbourne, C.1,
September 13, 1939.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 80, of September 21, 1939.

AUSTRALIAN MILITARY FORCES.

First Military District.

Australian Army Medical Corps.

Captain P. F. Murphy ceases to be seconded, 1st September, 1939.

Australian Army Medical Corps Reserve.

To be Honorary Captains—Peter Roylance Delamothe, Alan Robert Townsend, Alfred Eric Mason, Joseph Andrew Arratta, Cecil Thomas Underwood, Donald Ashley Billing Carter, Maurice Leslie Unwin, Thomas Felix Washington Power, Thomas Leo Cooney, Claude James Constable, Victor Richmond Woodhill, Clive William Taylor, Peter McLean Davidson, Harry Masel, Milton Geaney, Hugh Clement Murphy, Alexander Norman Park, Alexander Mayne Martell, Robert Lionel Rankin, Charles Maurice McCarthy and John King Joyce, 19th August, 1939; Horace William Johnson, Martin Joseph McKillop, Clive Lansdell Paine, John Cyril Thompson, Clarence Kingsley Haskard, Clive Gowan Williams, Ivo William Webster Charles, Alan Edward Lee, Leo Thomas Grattan Geraghty, James Vincent Guinane, Ronald Richmond Winton, Harold William Harbison, Felix Wilfrid Arden, Clive Wentworth Uhr, James Lee Selwood, William Dennis Ryan, James Cameron Hemsley, William Frederick Machin, Bruce Boyle Barrack, Keith Joseph Hill, John Edward Overstead and Alphonso Jean Gardiner Nicholson, 1st September, 1939.

Second Military District.

Australian Army Medical Corps.

Captain J. C. Loxton and Captains (provisionally) B. Jarvis, W. L. Fowles and C. A. C. Leggatt are brought on the authorized establishment, 1st August, 1939. Captain (provisionally) C. T. Petherbridge is borne supernumerary to establishment pending absorption, 1st August, 1939. Captain J. F. Lipscomb is transferred to the Reserve of Officers (A.A.M.C.), 1st September, 1939. Captain (provisionally) N. Joseph is transferred to the Reserve of Officers (A.A.M.C.) and to be Honorary Captain, 1st August, 1939.

Australian Army Medical Corps Reserve.

To be Honorary Lieutenants—William Theodore Winning and John Allan Clark, 14th August, 1939, and 21st August, 1939, respectively.

Third Military District.

Australian Army Medical Corps.

To be Captain (provisionally) supernumerary to establishment pending absorption—Thomas Harry Ackland, 1st September, 1939. Honorary Captain V. B. Brenton is appointed from the Reserve of Officers (A.A.M.C.), and to be Captain (provisionally) supernumerary to establishment pending absorption, 25th August, 1939. Major W. Davis is transferred to the Reserve of Officers (A.A.M.C.), 30th August, 1939.

Australian Army Medical Corps Reserve.

To be Honorary Captains—Keith Chisholm Ross and Douglas Arthur Carter, 22nd August, 1939; Cecil William John Rait, 24th August, 1939; John Holmes Shaw, 25th August, 1939, and Sydney Bernard Hudson, 28th August, 1939. *To be Honorary Lieutenant*—Walter Thomas Clemons Thomas, 28th August, 1939.

Fourth Military District.

Australian Army Medical Corps.

To be Major—Captain B. S. Hanson, 4th September, 1939. *To be Captain (provisionally)*—Sholto John Douglas, 29th August, 1939. Honorary Captain R. F. West is appointed from the Reserve of Officers (A.A.M.C.) and to be Captain (provisionally) supernumerary to establishment pending absorption, 30th August, 1939. Captain (provisionally) C. S. Swan is brought on the authorized establishment, 29th August, 1939.

Australian Army Medical Corps Reserve.

To be Honorary Captains—George David Harris, Laurance Llewellin Davey, Douglas Robson Wallman, Colin Gurner, Alfred Ladyman Tostevin and Michael Schneider, 30th August, 1939; Kenelm Charles Scutt, Malcolm Turner Cockburn and Owen Meredith Moulden, 4th September, 1939.

Fifth Military District.

Australian Army Medical Corps.

Major A. L. Dawkins is transferred from the Australian Army Medical Corps, 4th Military District, supernumerary to establishment pending absorption, 23rd August, 1939.

Australian Army Medical Corps Reserve.

To be Honorary Captains—Keith Forbes Abernethy and Richard Roderick Andrew, 23rd August, 1939.

Obituary.

JANE STOCKS GREIG.

We regret to announce the death of Dr. Jane Stocks Greig, which occurred on September 16, 1939, at Brighton, Victoria.

ALFRED ALEXANDER SMITH.

We regret to announce the death of Dr. Alfred Alexander Smith, which occurred on September 17, 1939, at Clare, South Australia.

HARRIETT ELIZA BIFFIN.

We regret to announce the death of Dr. Harriett Eliza Biffin, which occurred on September 22, 1939, at Lindfield, New South Wales.

Nominations and Elections.

The undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Macourt, Henry Ross, M.B., B.S., 1938 (Univ. Sydney), 54, Roseville Avenue, Roseville.

Books Received.

PROCTOLOGY FOR THE GENERAL PRACTITIONER, by F. C. Smith, M.D., M.Sc., F.A.P.S.; 1939. Philadelphia: F. A. Davis Company. Medium 8vo, pp. 390, with 142 illustrations and 3 coloured plates. Price: $4.50 net.

Diary for the Month.

OCT. 3.—New South Wales Branch, B.M.A.: Council (Quarterly).
OCT. 4.—Victorian Branch, B.M.A.: Branch.
OCT. 4.—Western Australian Branch, B.M.A.: Council.
OCT. 5.—South Australian Branch, B.M.A.: Council.
OCT. 6.—Queensland Branch, B.M.A.: Branch.
OCT. 10.—New South Wales Branch, B.M.A.: Executive and Finance Committee, Organization and Science Committee.
OCT. 13.—Queensland Branch, B.M.A.: Council.
OCT. 17.—New South Wales Branch, B.M.A.: Ethics Committee.
OCT. 18.—Western Australian Branch, B.M.A.: Branch.
OCT. 19.—New South Wales Branch, B.M.A.: Clinical Meeting.
OCT. 24.—New South Wales Branch, B.M.A.: Medical Politics Committee.
OCT. 25.—Victorian Branch, B.M.A.: Council.
OCT. 26.—New South Wales Branch, B.M.A.: Branch.
OCT. 26.—South Australian Branch, B.M.A.: Branch.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenencies* sought, etc., see "Advertiser", pages xvi-xviii.

GOVERNMENT OF NORTH BORNEO: Medical Officer.
ST. GEORGE DISTRICT HOSPITAL, KOGARAH, NEW SOUTH WALES: Honorary Consulting Anæsthetists, Senior Resident Medical Officer.
THE HORNSBY AND DISTRICT HOSPITAL, HORNSBY, NEW SOUTH WALES: Honorary Officers.
THE UNIVERSITY OF ADELAIDE, SOUTH AUSTRALIA: Director in Obstetrics.
VICTORIAN EYE AND EAR HOSPITAL, MELBOURNE, VICTORIA: Resident Medical Superintendent, Resident Surgeons.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 178, North Terrace, Adelaide.	All Lodge appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	Wiluna Hospital. All Contract Practice Appointments in Western Australia.

Editorial Notices.

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